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THE SYNDROME OF BERNHEIM

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THE clinical picture of right heart failure is routinely ascribed to dilatation of the right cardiac chambers. Thus, when systemic venous engorgement occurs in the hypertensive or arteriosclerotic subject, it is presumed that the right ventricle, having been unable to withstand the burden imposed by left ventricular insufficiency, has dilated and failed. That this is not always the case was demonstrated in 1908 by Bernheim who found stenosis rather than dilatation of the right ventricle in a large number of patients who had died with the classic symptomatology of right heart failure. In these cases he observed that encroachment upon the cavity of the right ventricle had resulted from deviation of a thickened interventricular septum which, in some instances, almost approximated the lateral wall of this chamber. It was evident from his findings that hypertrophy and dilatation of the left ventricle had materially reduced the capacity of the right ventricle through the mechanism of a bulging, interventricular septum. In some cases, the author found the latter chamber so compromised in its apical half that it appeared as a narrow slit between the bulging septum and the lateral wall with the result that only the upper half of the right ventricular cavity and the pulmonary conus remained open.

In 1910 and again in 1915, Bernheim[1, 2] emphasized that the venous engorgement so characteristically observed in these subjects, is a direct consequence of obstruction to blood flow through the right ventricle and not, as is usually supposed, a manifestation of right heart failure. He pointed out that stenosis produced by septal bulging could be diagnosed during life if careful attention is paid to the evolution of symptoms. Thus, while the majority of cardiac patients with left ventricular hypertrophy manifest visceral congestion and edema only after dyspnea and pulmonary congestion have been present for some time, the former symptoms are the first to appear when stenosis of the right ventricle has supervened. Consequently, the diagnosis of Bernheim's syndrome rests upon the clinical picture of isolated right heart failure in association with left ventricular hypertrophy. These interesting observations, however, were forgotten until Laubry and his associates,[3] having studied patients with similar signs, proposed, in 1924, a completely different interpretation.

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They contended that, since the picture of right heart failure is present in cases where the right ventricle is not dilated but narrowed, all instances of cardiac decompensation must actually be instances of left heart failure. Other students of the subject, however, challenged this point of view and supported Bernheim's original observations and conclusions. The publications of Martini and Joselevich,[4, 8, 10, 17-19] Bullrich,[5, 6] Mayer and Mazzei,[7, 9] Mazzei,[11-15, 21, 22] and others characterized the syndrome as possessing sufficient clinical and anatomic individuality to warrant its acceptance as an entity in clinical medicine.

In 1930, Mazzei,[11] on the basis of his own observations and those reported by others, divided the clinical picture of Bernheim's syndrome into two periods. The first, or anatomic period, is of variable duration and does not present important clinical signs. Interference with the filling of the right ventricle is counterbalanced by dilatation of the infundibular portion of the chamber and by enlargement of the right auricle. The second, or clinical period, has a shorter evolution and is itself divided into two stages. In the first stage, the signs of venous obstruction due to right ventricular stenosis become manifest. The pulmonary blood flow remains undisturbed, so that there is a dissociated failure of the circulation. In the second stage, which is usually terminal, stasis of the lesser circulation is added to the symptoms of systemic venous engorgement. Mazzei[21] believed that the terminal appearance of pulmonary congestion in these cases signified failure of the right ventricle. Martini and Joselevich,[17] however, took issue with this view. They maintained that failure of the lesser circulation is not observed in the first period of the syndrome because the dilatation of the left ventricle, which provokes deviation of the septum, is active and not passive. In the latter part of the second period, the appearance of râles at the lung bases presents evidence of passive dilatation and true failure of the left ventricle. Inasmuch as this end stage represents total failure of the heart, it is only the earlier period, according to Martini and Joselevich, which can be recognized as Bernheim's syndrome.

Although numerous papers on this subject have appeared in the French, Italian, Spanish, and Latin American literature,[1-36] a careful search of the *Index Medicus* has revealed only one case report[37] in the English language. The syndrome appears to have attracted little attention from physicians in this country. Fishberg,[38] in his textbook, *Heart Failure*, refers to the syndrome and states that he has observed many necropsies which support Bernheim's views. White,[39] in the latest edition of his book, mentions the syndrome for the first time but expresses doubt on theoretical grounds, as to its entity.

The purpose of this paper, therefore, is to present our findings in three cases of Bernheim's syndrome, two of which were diagnosed during life and confirmed at necropsy and one discovered unexpectedly on post-mortem examination.

CASE REPORTS

CASE 1.—C. C., a 44-year-old Filipino seaman was first admitted to the U. S. Marine Hospital on Oct. 24, 1939, for treatment of asymptomatic hypertension which disqualified him for employment. His past history revealed that he contracted gonorrhea in 1928 and that he had had no other illnesses except the usual childhood diseases.

Examination revealed a well-developed and well-nourished adult male of small stature, in no acute distress. His temperature was 98.6° F., pulse, 94, and respirations, 18. The fundi showed disc margins which were clear and distinct. The retinal arterioles were tortuous and "silver-wire" in character. Arteriovenous notching was evident. There were no retinal hemorrhages or exudates. The radial pulses were of good volume, equal, and regular. The blood pressure was 234/142. There was engorgement of the cervical veins.

The apex impulse of the heart was 2.5 cm. outside the left mid-clavicular line in the fifth intercostal space. The sounds were of good quality and no murmurs were heard. The second aortic sound was loud and snapping. The lungs were resonant throughout. The breath sounds were vesicular in type and there were no râles. The liver and spleen were not palpable. There was no dependent edema. The circulation time (arm to tongue) was 19 seconds using decholin. The venous pressure was 160 mm. of water. Blood Wassermann and Kahn reactions were negative. Routine urine analysis showed specific gravity of 1.015 and a trace of albumin. X-ray of the heart revealed the maximum cardiac measurements as 5 cm. to the right and 10.5 cm. to the left; the transverse diameter of the chest was 28.4 centimeters. The lateral view showed obliteration of the retrocardiac space. Electrocardiogram revealed normal sinus rhythm with a ventricular rate of 86; P-R interval, 0.16 second; QRS interval, 0.08 second; slurred QRS in limb leads; T waves inverted in Leads II and III; and right axis deviation.

After one month's hospitalization, the blood pressure showed little change, and the patient was discharged. He was readmitted to the hospital on May 16, 1940, with the complaint of swelling of the abdomen and legs. There was no history of exertional or paroxysmal nocturnal dyspnea.

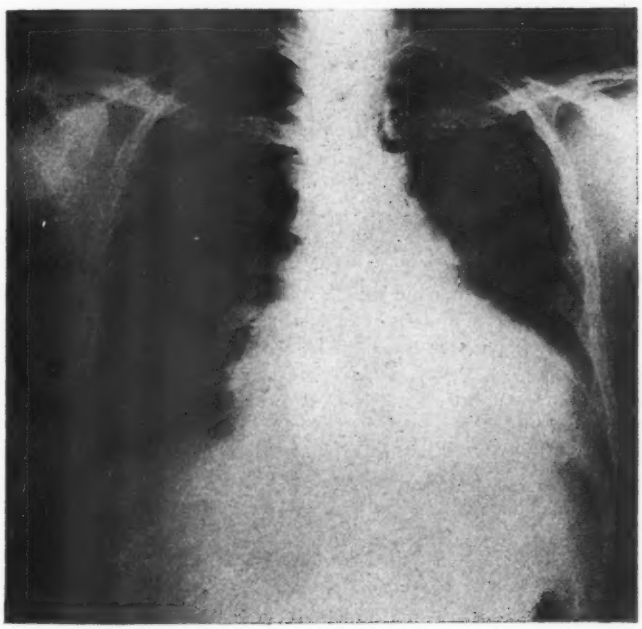

Fig. 1.

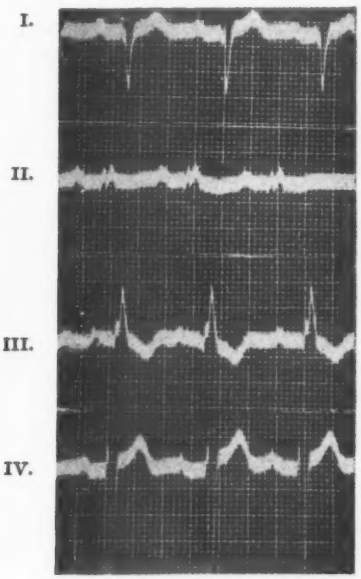

Fig. 2.

Fig. 1.—Case 1. Roentgenogram showing marked enlargement of the heart. Note the absence of pulmonary congestion.

Fig. 2.—Case 1. Electrocardiogram showing right axis deviation.

Examination revealed a blood pressure of 204/140. The left border of the heart was in the anterior axillary line. No murmurs were heard. The heart rate was 80 per minute and regular. Scattered rhonchi were present throughout the lungs. There was marked distention of the entire abdomen and a fluid wave was elicited. On percussion, there was dullness in the flanks as well as shifting dullness on change of position. There was moderate edema of the scrotum and marked pitting edema of the legs as high as the mid-thigh region. The arm-to-tongue circulation time was 20 seconds. Roentgenogram of the heart (Fig. 1) showed an appreciable increase in the transverse diameter of the cardiac silhouette as compared with that of the former admission. Maximum cardiac measurements were 5.6 cm. to the right and 12.4 cm. to the left; the transverse diameter of the chest was 27.5 centimeters. There was no evidence of pulmonary congestion. The electrocardiogram was essentially the same as on first admission showing right axis deviation and inversion of T waves in Leads II and III (Fig. 2). Urinalysis showed a specific gravity of 1.018 with no

abnormal findings. The blood picture revealed 3,500,000 red blood cells and 70 per cent hemoglobin. The nonprotein nitrogen of the blood was 46 mg. per cent; the total serum protein was 6.9 Gm. per cent; albumin was 3.5 Gm. per cent and globulin, 3.4 Gm. per cent. Icterus index was 15.

The patient was digitalized and 2 c.c. of Mercupurin were administered intravenously every three to four days. On this therapy together with fluid and salt restriction, dependent edema and ascites disappeared. At the time of discharge, on July 8, 1940, the blood pressure was 185/140, the pulse was 80 per minute, and there was no evidence of circulatory failure except for prominence of the cervical veins.

The patient was readmitted to the hospital Oct. 1, 1940, with the same complaint; namely, marked swelling of the lower extremities and abdominal distention. There was also slight dyspnea on moderate exertion. In the interval, he had been on a maintenance dosage of digitalis.

Physical examination revealed no evidence of dyspnea or orthopnea. The patient was able to lie flat in bed without respiratory distress. The lips were cyanotic. There was marked distention of the cervical veins. The heart findings were as formerly noted. Blood pressure was 200/140. There were no murmurs or thrills. The cardiac rate was 98 per minute and regular. There was marked prominence of the abdominal wall with eversion of the umbilicus. The liver border was 3 fingerbreadths below the right costal margin in the mid-clavicular line. A fluid wave was elicited in the abdomen and there was shifting dullness on change of position. There was scrotal edema and swelling of the lower extremities as high as the mid-thigh region. The arm-to-tongue circulation time was 25 seconds. The venous pressure was 180 mm. of water. X-ray of the heart showed no further change in cardiac size. There was still no evidence of pulmonary congestion. The electrocardiogram was as formerly noted. The total serum protein was 6 Gm. per cent with an albumin level of 3.4 Gm. per cent and a globulin level of 2.6 Gm. per cent.

Four thousand cubic centimeters of clear, yellow fluid were withdrawn by abdominal paracentesis. The maintenance dosage of digitalis was increased to 1½ grains twice daily and 2 c.c. of mercupurin were given every two to three days. It was found that, in spite of the restriction of fluid and sodium, edema would develop rapidly if the mercurial diuretic was withheld for more than several days. Gain in weight was used as the indication for mercupurin therapy. The patient was maintained on this regime for seven months and was again discharged on a maintenance dosage of digitalis. He returned as an out-patient once each week for an injection of mercupurin but, after two months, had to be readmitted to the hospital because of a recurrence of symptoms.

Upon entering the hospital on June 3, 1942, there was no evidence of respiratory distress. Blood pressure was 182/136. The pulse was 96 per minute and regular except for an occasional ectopic beat. Cyanosis of the face was marked. The cervical veins were distended and pulsating. The heart was markedly enlarged to the left and there was a soft blowing systolic murmur at the apex as well as a protodiastolic gallop. The lungs were clear on auscultation except for a few moist râles at both bases posteriorly. There was marked protrusion of the abdomen. The liver was 3 fingerbreadths below the right costal margin in the mid-clavicular line. Marked pitting edema of the lower extremities and scrotum was present. The arm-to-tongue circulation time was 40 seconds using decholin. The venous pressure was 200 mm. of water. Roentgenogram of the heart showed marked enlargement in the transverse diameter and also toward the back. The maximum cardiac measurements were 6.8 cm. to the right and 10.8 cm. to the left; the transverse diameter of the chest was 25.3 centimeters. There was obliteration of the retrocardiac space. There was still no evidence of pulmonary congestion. Electrocardiogram showed normal sinus rhythm with a ventricular rate of 80; P-R interval, 0.21 second; QRS interval, 0.09 second; slurred QRS in limb leads; T waves diphasic in Lead II and inverted in Lead III; and right axis deviation.

The patient showed slight improvement at first on rest in bed and injections of mercupurin, but, after several months, his condition became steadily worse and dyspnea and orthopnea became evident. The latter symptoms thus appeared for the first time after three years of observation. At the same time, the systolic apical murmur increased in intensity and duration. Moist râles were heard at the lung bases posteriorly and x-ray of the chest revealed pulmonary congestion involving both lower lobes. Ascites and dependent edema persisted. The patient failed to respond to all measures and died on Oct. 2, 1942. The final medical impression was hypertensive heart disease associated with Bernheim's syndrome.

Autopsy.—External Examination: The body was that of a male adult Filipino of small stature. There was pitting edema of the ankles and legs. The abdomen was protuberant. The mucous membranes were pale and showed no evidence of icterus. There were no petechiae.

Internal Examination: The peritoneal cavity contained blood-tinged, yellow fluid amounting to approximately 3 liters. There were adhesions between the omentum and the anterior abdominal wall suggesting the healing of previous sites of paracenteses. The pleural cavities showed no free fluid present. There were no adhesions on the left side but a few fibrous attachments were found on the right. The pericardial cavity had a smooth lining with a content of about 25 c.c. of clear fluid. The heart was greatly enlarged, particularly to the left, and weighed 710 grams. A transverse section a short distance above the apex of the heart revealed a remarkable thickening of the interventricular septum which ranged from 2 to 2.5 cm. in thickness and which had a firm, resilient texture. The right ventricular cavity was encroached upon by this convex septal bulging. However,

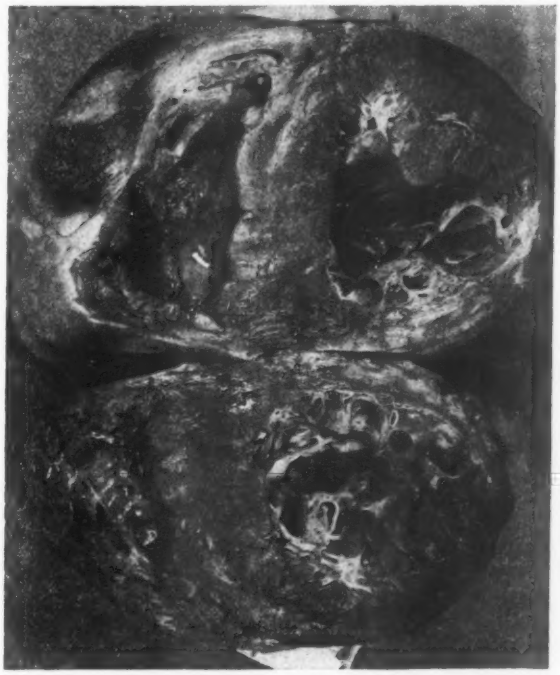

Fig. 3.—Case 1. Transverse section of the heart showing marked hypertrophy of the left ventricle and the interventricular septum. The right ventricle is reduced in size by the bulging septum, the left ventricle by concentric hypertrophy.

the capacity of the right ventricle appeared to be about equal to that of the left, since the latter was markedly reduced by concentric hypertrophy of the myocardium (Fig. 3). There was a considerable area of fibrous replacement of the left ventricular wall laterally, extending from the mid-portion to include the apex. Dissection of the coronary arteries showed that the left was predominant. The caliber was large. There were numerous atheromatous foci. Fresh thrombus could not be found. The valves of the heart showed considerable atheroma. The dimensions of the valves were: mitral circumference, 13 cm.; aortic, 6.5 centimeters. There was no evidence of old or recent vegetation, fibrosis, or calcification. The right auricle was considerably dilated. No thrombi were present. The aorta showed considerable thickening of the entire wall. There was no aneurysm. The intima showed longitudinal wrinkles and flat grayish-white foci suggesting syphilitic aortitis. The lungs were partially aerated and had a brownish-yellow color indicative of passive congestion. On section the tissue was moist and considerable hemorrhagic fluid exuded from all lobes. Small foci appeared to be recently consolidated. These were distributed centrally in both lungs. The liver weighed 1,100 grams. Its capsule was smooth, and, on section, the substance was yellow-brown and not unduly resistant. The principal vessels appeared normal. The spleen was not enlarged and showed normal markings.

Gastrointestinal Tract: Numerous areas of intense congestion were found in the stomach and small intestine. There was marked thickening and hemorrhagic infiltration in the descending colon for about 25 cm. suggesting thrombosis of the veins. The kidneys weighed 305 grams together and were of about equal size. On section each kidney showed a cortical width of about 7 millimeters. On stripping the capsule, the surface was left finely granular. In addition there were a number of small sunken and retracted areas of fibrosis.

Microscopic Examination.—The myocardial fibers were larger than usual. There was a moderate amount of interstitial connective tissue interrupting the muscle fibers over an extent of several low-power fields. Dilated capillary vessels were seen in the middle of these scars. Large branches of the coronary system were seen in section and showed very prominent atheromatous thickening of the intima and severe reduction of the lumen. The aorta showed numerous small scars in the media together with dilated capillary vessels and clusters of lymphocytes. There were also foci of vacuolization and lipoid deposit in the intima. The lungs showed marked congestion of the capillaries and veins throughout several sections. The alveolar spaces contained many large monocytes containing brown granular pigment. There were small areas of infarction. The general architecture of the liver was well preserved, the central areas were congested. Most of the glomeruli of the kidneys were well preserved, but a few scattefed, partly or completely fibrotic glomeruli were noted. Numerous interstitial areas appeared fibrosed and moderately infiltrated by lymphocytes. The arteries showed severe thickening and hyalinization involving large and small branches. Necrosis of arterial walls could not be found. Pathologic diagnoses were: fibrosis of the myocardium; hypertrophy of the heart; coronary arteriosclerosis; nephrosclerosis; passive congestion of the lungs, liver, and kidneys; syphilitic aortitis; anasarca; and generalized arteriosclerosis.

Case 2.—F. R., a 42-year-old Filipino merchant seaman was admitted to the hospital on Sept. 27, 1943, with a history of persistent swelling of the ankles since 1939, followed, a short time later, by shortness of breath on exertion. One week prior to admission he had some discomfort in the chest and because of this was referred to the hospital. The past history was essentially negative except for a Neisserian infection in 1918.

Physical examination revealed a well-nourished and well-developed male of small stature, not acutely ill. There was no evidence of dyspnea or orthopnea. Both pupils reacted to light and accommodation. The fundi showed marked tortuosity of the vessels and hyperemic discs. No hemorrhages were noted. The lungs were clear to percussion and auscultation except for a few fine râles at both bases. The heart was considerably enlarged with the left border in the anterior axillary line. There was a blowing systolic murmur at the apex. The aortic second sound was louder than the pulmonic. The cardiac rate was 114 per minute; the rhythm was regular. Blood pressure was 230/155. The liver edge was felt 3 fingerbreadths below the right costal margin in the mid-clavicular line. There was evidence of free fluid in the abdomen. There was moderate pitting edema of both ankles. The arm-to-tongue circulation time was 20 seconds using decholin. The venous pressure was 150 mm. of water. A diagnosis of hypertensive heart disease with Bernheim's syndrome was made. X-ray examination of the heart showed the cardiac shadow to be markedly enlarged (Fig. 4). The maxium cardiac measurements were 5.9 cm. to the right and 11.6 cm. to the left; the transverse diameter of the chest was 26 centimeters. A lateral study showed marked obliteration of the retrocardiac space by the left ventricle. Fluoroscopic examination revealed definite left ventricular enlargement but failed to show any evidence of right auricular dilatation. Electrocardiogram before the administration of digitalis, showed normal axis with inversion of the T waves in the limb leads (Fig. 5). Urinalysis showed a specific gravity of 1.010 with moderate albuminuria, many red blood cells, and a few hyaline and granular casts. The blood count was essentially normal. Wassermann and Kahn tests were negative. Nonprotein nitrogen was 60 mg. per cent. Total protein was 7.2 Gm. per cent with an albumin level of 3.3 Gm. per cent and a globulin level of 3.9 Gm. per cent.

While in the hospital the patient showed little evidence of dyspnea. There were a few moist râles at both lung bases. Moderate edema of the lower legs persisted. Patient was digitalized and given mercupurin intravenously once or twice weekly with some improvement. On Nov. 18, 1943, the blood pressure was 250/160. There was a loud systolic murmur and gallop rhythm at the apex. The heart rate was 80 per minute and the rhythm

was regular. The second pulmonic sound was markedly accentuated and considerably louder than the second aortic sound. The lungs showed many moist râles bilaterally. The circulation time, arm-to-tongue, was 65 seconds. The venous pressure was 270 mm. of water. Dyspnea and orthopnea were present. On Nov. 30, 1943, the patient developed a convulsive seizure lasting several minutes. Convulsions recurred in rapid sequence and the patient lapsed into coma and expired Dec. 4, 1943.

Autopsy.—External Examination: The body was that of a normally developed and fairly well-nourished oriental male of adult age. The mucous membranes showed no evidence of icterus. There were no petechiae.

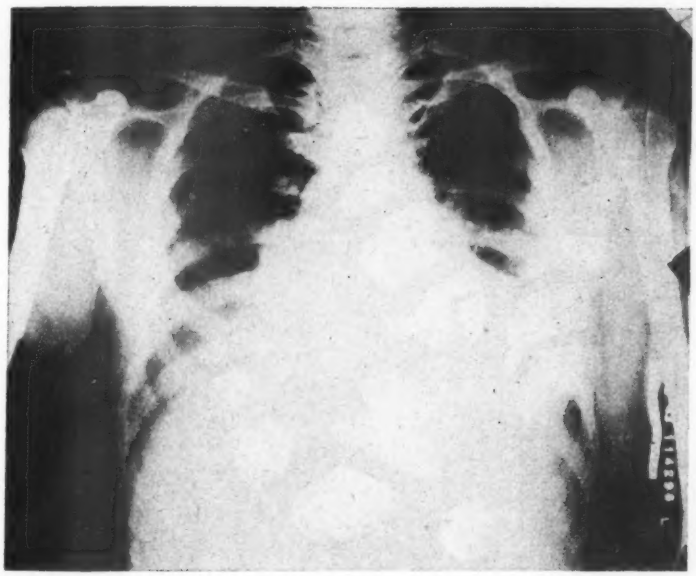

Fig. 4.

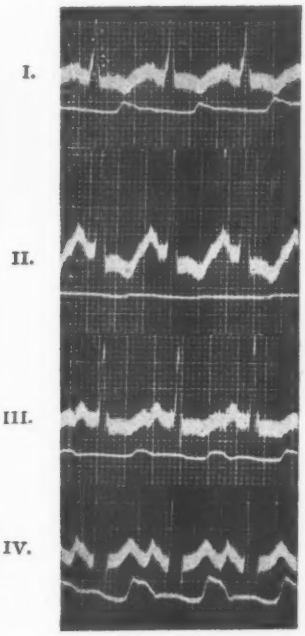

Fig. 5.

Fig. 4.—Case 2. Roentgenogram showing marked enlargement of the heart.
Fig. 5.—Case 2. Electrocardiogram showing normal electrical axis.

Internal Examination: The peritoneal cavity contained no free fluid or exudate. The pleural cavities showed a little free fluid present. There were no adhesions. The pericardial cavity was found to have a lining which was dull and fibrinous with no free fluid noted. The heart weighed 725 grams. The surface was fibrinous with hemorrhagic foci posteriorly. The myocardium was firm in consistency and showed no foci of softening or hemorrhage. The thickness of the left ventricle was 2.3 centimeters. The septum was 3 cm. thick. The right ventricular cavity was markedly compromised by the hypertrophied and bulging septum (Figs. 6 and 7). The coronary arteries showed patent orifices. On dissection they were patent and had many foci of atheroma and calcification. The endocardium showed no mural thrombi. The valves had normal surfaces. No vegetations were observed on any of the valves. The measurements were: tricuspid, 10 cm.; mitral, 9 cm.; pulmonary, 7 cm.; and aortic, 7.2 centimeters. The aorta showed slight patchy yellow discoloration and a few atheromatous plaques. The lung surfaces were smooth, and there was no exudate. On section, the tissue contained air in all portions and had a yellowish color. The liver weighed 850 grams. Its surface was smooth; it was firm in texture; and it was mottled in color. The spleen was not enlarged. On section its pulp was purple in color and firm in consistency. The kidneys were symmetrical and weighed 400 grams together. On section the cortex had a width of 7 millimeters. The capsule stripped readily, leaving a finely granular surface. The markings were unclear and the color was purple.

Microscopic Examination.—The large coronary trunks had thick hyaline walls with foci of lipoid and lymphocytes. The epicardium was covered by red cells and fibrin in certain sections. The myocardial fibers were greatly enlarged and the nuclei huge. Some fibrosis was apparent. The glomeruli of the kidneys contained blood cells. There were

partly hyalinized and swollen areas especially close to the afferent artery or actually involving it. Peculiar blotchy hyaline necrosis thickened and occluded many arterioles. The larger arteries showed severe atheromatous changes. The centrolobular regions of the liver were heavily infiltrated by red blood cells. The arterioles showed necrotic hyaline areas. There were many pigment-laden phagocytes in the air spaces of the lungs. Many bronchioles were filled with polynuclear cells. Hyaline thrombi of the arterioles were noted. The pathologic diagnoses were: nephrosclerosis, malignant; diffuse arteriolitis; and hypertrophy of the heart, especially the septal portion of the left ventricle.

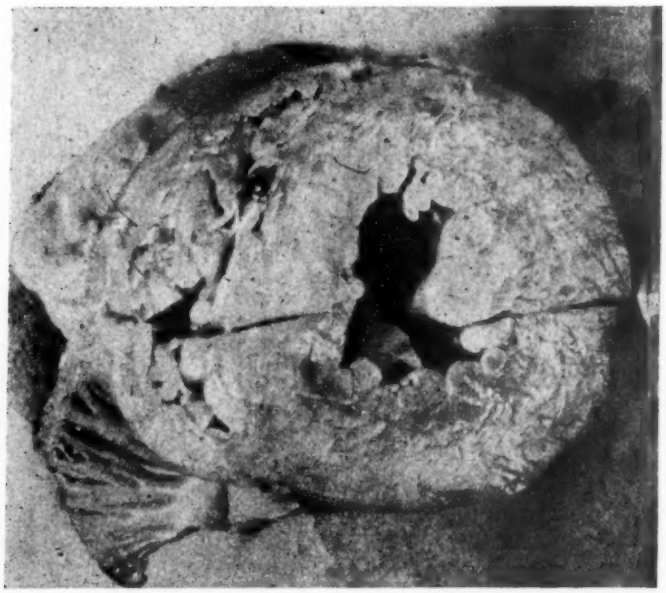

Fig. 6.—Case 2. Transverse section of heart (midway between apex and base) showing marked left ventricular hypertrophy. The cavity of the right ventricle is greatly reduced in size by the bulging interventricular septum which measures 3 cm. in thickness.

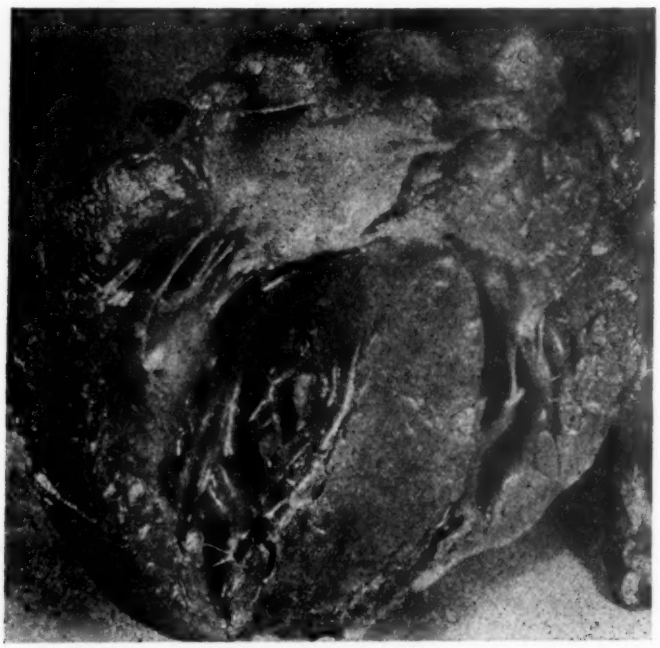

Fig. 7.—Case 2. Longitudinal section of the heart showing relative size of the ventricles. Note the bulging septum and the reduced capacity of the right ventricle.

CASE 3.—W. R., a 73-year-old white merchant seaman was admitted to the hospital on Sept. 22, 1943, with the complaint of dyspnea on exertion and swelling of the ankles of two months' duration. Two weeks prior to admission he experienced a severe attack of dyspnea requiring an injection of morphine. Past history was noncontributory. There was no previous history of rheumatic fever or syphilis.

Physical examination revealed an elderly white male not acutely ill. His temperature was 98.6° F., and his respirations, 20. The face and nail beds were cyanotic. The blood pressure was 130/80. There was marked distention of the cervical veins. The left border of the heart was 3 cm. outside the left mid-clavicular line. There was a loud, harsh, systolic murmur over the precordium with maximum intensity at the aortic area where a systolic thrill was palpated. The second aortic sound was absent. A mid-diastolic rumble was heard at the apex. The cardiac rhythm was irregularly irregular with an approximate ventricular rate of 88 per minute. The lungs were clear throughout; no râles were heard. The liver border was 4 fingerbreadths below the right costal margin in the mid-clavicular line. There was marked pitting edema involving the legs as high as the mid-thigh region. Large blebs containing clear fluid, were present over the lower legs. Blood Wassermann and Kahn reactions were negative. Routine urinalysis showed a specific gravity of 1.015 with negative findings. X-ray of the heart revealed the maximum cardiac measurements as 4.8 cm. to the right and 12.6 cm. to the left; the transverse diameter of the chest was 33.2 centimeters. There was evidence of small infarcts in the right lung base adjoining the heart (Fig. 8). The lateral view showed calcification in both the mitral and aortic valve rings. Electrocardiogram revealed auricular fibrillation with a ventricular rate of approximately 60; diphasic T waves in the limb leads due to digitalis effect; and normal axis (Fig. 9).

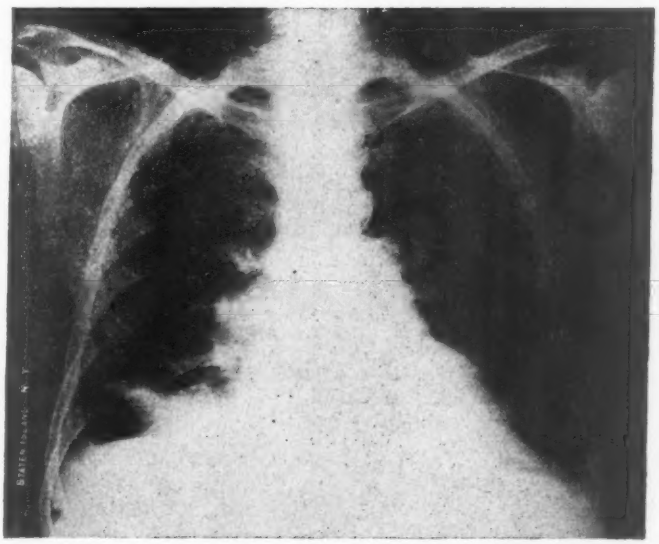

Fig. 8.—Case 3. Roentgenogram showing marked enlargement of the heart. Note absence of pulmonary congestion. There are small infarcts at the right lung base.

The clinical diagnosis was rheumatic heart disease, mitral and aortic stenosis, auricular fibrillation, and congestive heart failure. The patient was digitalized and was given mercupurin intravenously twice weekly. While in bed he manifested little dyspnea but continued to show the signs of marked right heart failure. He was discharged on Jan. 14, 1944, for continued rest in bed at home.

The patient was readmitted on Feb. 17, 1944, with the complaint of marked swelling of the lower extremities which were covered with "blisters" in their lower parts.

Examination revealed similar findings as on previous admission. Cyanosis was marked. There was engorgement of the cervical veins. Moist râles were heard at both lung bases posteriorly. The abdomen was distended and a fluid wave was elicited. There was marked pitting edema of both lower extremities as high as the groin. Large weeping ulcers were present over the middle two-thirds of both tibiae.

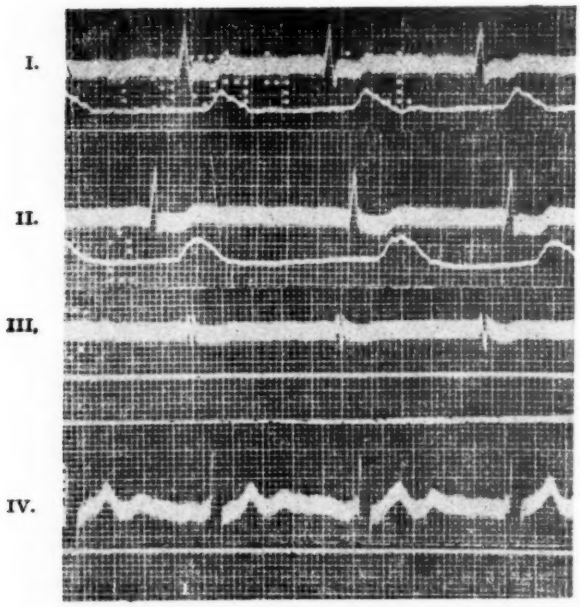

Fig. 9.—Case 3. Electrocardiogram showing normal electrical axis.

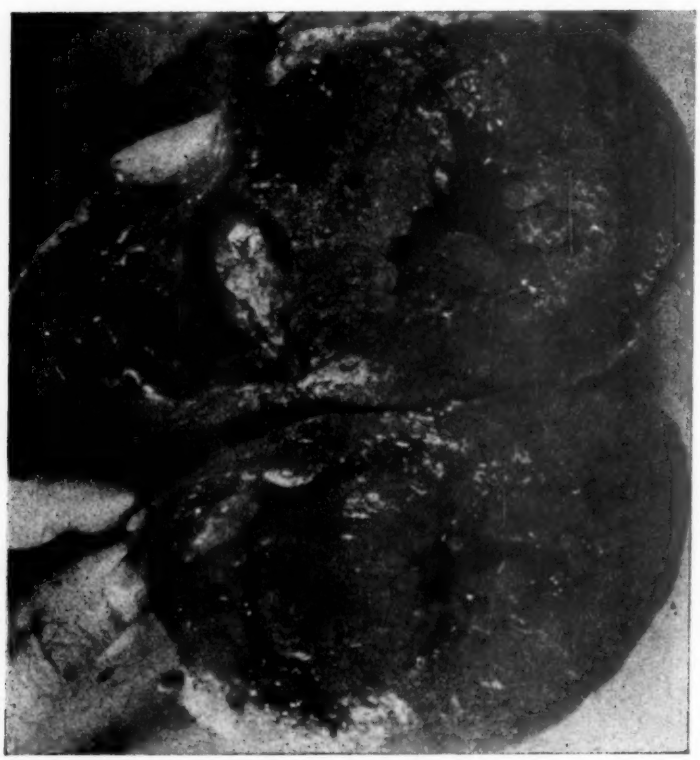

Fig. 10.—Case 3. Transverse section of the heart (midway between apex and base). Note convex bulging of the septum which measures 3.2 cm. in thickness. The right ventricle is markedly reduced in size.

On rest in bed, digitalis, and mercupurin, slight improvement ensued. Later, the patient became drowsy and appeared dehydrated. A blood nonprotein nitrogen was found to be 138 mg. per cent. All further measures failed to influence the course of the illness, and the patient expired on March 19, 1944.

Autopsy.—External Examination: The body was that of a well-developed and well-nourished white man of advanced age. There was pitting edema of the legs. The mucous membranes showed no evidence of icterus. There were no petechiae.

Internal Examination: The peritoneal cavity had some free fluid but no exudate. There were no adhesions. The pleural cavities showed smooth surfaces and a little free fluid was present. The pericardial cavity had a smooth lining and contained about 25 c.c. of clear fluid. The heart weighed 800 grams. The myocardium was firm in consistency and showed a few foci of flattening and retraction, especially in the right ventricle. The left ventricle measured 2.6 cm. in thickness. The interventricular septum bulged convexly toward the right, markedly encroaching upon the right ventricular cavity. The septum measured 3.2 cm. in thickness (Fig. 10). The coronary arteries showed small orifices. On dissection they were patent and had many foci of atheroma and calcification. The endocardium of the auricles showed friable pink mural thrombi. The valves had normal surfaces and tendons on the right side of the heart. The mitral curtains and tendons were short and adherent. There was marked deformity of the mitral and aortic valves with abundant knoblike calcified encrustations in the sinuses of Valsalva, holding the aortic orifice in a closed position.

The lung surfaces were dull and showed a fibrinous exudate. On section the tissue contained air in all portions except for large wedges of dark red consolidation sharply outlined and present in all lobes on both sides. The liver weighed 1,520 grams and showed a finely nodular surface. Its texture was firm and its color brown gray. The spleen was of normal size and on section its pulp was firm. The kidneys were about equal in size and weighed about 390 grams together. On section the cortex had a width of 6 millimeters. The capsule stripped easily, leaving a smooth surface. The markings were clear with a few small cysts and a rare zone of yellowish color with a reddened margin.

Microscopic Examination.—The myocardium showed considerable interstitial fibrosis. Many of the muscle fibers were enlarged. There was hyalinization with foci of calcification in the base of a valve. A few cellular foci were noted near blood vessels and beneath the endocardium. They consisted of large cells with ovoid nuclei. Some pigment was present. Large areas of necrosis and monocytic infiltration were noted in the wall of an auricle. Its lumen was occupied by a fibrin thrombus with masses of leucocytes and red blood cells enclosed. A large branch of the pulmonary artery had a degenerated edematous and fibrinoid wall with destruction of the endothelium. The lumen was filled by a thrombus of fibrin and blood cells with large central areas of laking. The small arteries of the lung also contained thrombi and showed foci of atheroma, edema, lamellar splitting, and monocytic infiltration. The alveoli contained a great many pigment-filled monocytes. The centrolobular areas of the liver were severely damaged and infiltrated by red blood cells. About half of the trabeculae were thus involved. Many phagocytes and some fibroblasts were noted. The arteries of the kidney were thick and hyaline. The great majority of the glomeruli and tubules appeared intact. Pathologic diagnoses were: rheumatic endocarditis with mitral and aortic stenosis; mural thrombosis of the auricles; pulmonary infarctions; arteriosclerosis of the pulmonary arteries; and central necrosis of the liver.

DISCUSSION

The advent of systemic venous engorgement as the first sign of circulatory embarrassment and its persistence in the absence of pulmonary congestion, suggested the clinical diagnosis of Bernheim's syndrome in Cases 1 and 2. Although ascites, enlargement of the liver, and dependent edema existed for some time, roentgenograms of the chest as well as physical signs failed to reveal evidence of pulmonary congestion until a later stage. The patients, moreover, were able to lie flat in bed and to engage in slight to moderate activity without manifesting respiratory distress. Correlated with these features of the clinical picture was the interesting observation in the early stage of the disease, of an elevated venous pressure with a comparatively normal arm-to-tongue circulation time. Interference with the filling of the right ven-

tricle, therefore, seemed more competent to explain these findings than did failure of this chamber following left ventricular insufficiency. Furthermore, if the engorgement of the systemic veins had had its pathogenesis in true insufficiency of the right ventricle, an appreciable dilatation of that chamber should have been noted. On the contrary, necropsy study revealed no dilatation of the right ventricle in spite of long standing symptoms of right heart failure. This finding, together with the marked displacement and bulging of the interventricular septum into the right ventricle, lends support to Bernheim's contention that interference with the filling of the right ventricle underlies systemic venous engorgement in these cases.

The development of pulmonary congestion as a terminal event in this syndrome, has been a subject of considerable discussion. In Case 1, a loud, blowing apical, systolic murmur and gallop rhythm developed with the onset of pulmonary congestion late in the course of the disease. In Case 2, gallop rhythm and marked accentuation of the second pulmonic sound similarly preceded the development of bilateral basal râles and orthopnea. It appears, therefore, that prior to these findings the clinical picture was dominated by signs and symptoms arising from obturation of the right ventricle; whereas, left ventricular failure, if it existed at all, was either minimal or neutralized by diminished right ventricular output. The normal circulation time and increased venous pressure in both cases, strengthen this view. With the appearance of gallop rhythm and relative mitral insufficiency, congestion of the lungs soon developed as a result of failure of the left ventricle.

In Case 3 in which the cardiac lesions were aortic and mitral stenosis, the anatomic features of Bernheim's syndrome were discovered unexpectedly at necropsy. In retrospect, the only unusual feature was the extreme degree of "right heart failure" associated with minimal pulmonary congestion. Inasmuch as a similar clinical picture is sometimes seen in mitral stenosis with failure of the right ventricle, no other mechanism was suspected. The discovery of a greatly reduced right ventricular chamber at autopsy was, therefore, an interesting paradox.

Certain electrocardiographic findings have been described as characteristic of Bernheim's syndrome. Some authors have stated that left ventricular preponderance is the usual feature of the electrocardiogram. From anatomic considerations, one would indeed anticipate rotation of the electrical axis of the heart to the left; nevertheless, in none of the three cases which we have presented, was this finding observed. On the contrary, there was right ventricular preponderance in Case 1 and a normal electrical axis in Cases 2 and 3. Glushein and Geer[37] similarly reported "no axis deviation" in the case which they observed. These seemingly paradoxical findings require explanation in view of the marked difference in the relative size and thickness of the respective ventricles. Inasmuch as Eliaser and Konigsberg[40] found right axis deviation in 40 per cent of their cases of cardiac aneurysm, Rowland[41] regards right axis deviation in association with left ventricular enlargement as suggestive of this diagnosis. Klainer,[42] in a recent report, found only thirty-six instances of right axis deviation in hypertensive or arteriosclerotic heart disease in all the electrocardiographic records of the Beth Israel Hospital over a ten-year period. He attributed the right axis deviation to widespread necrosis of the left ventricle which nullified the effects of hypertrophy of this chamber. It also seems possible, however, that a bulging and deviated interventricular septum, as seen in the syndrome of Bernheim, may alter the electrical axis of

the heart and even be responsible for unexpected right axis deviation in hypertensive heart disease.

Dulcos[28] regarded the kymographic demonstration of right auricular dilatation as a valuable diagnostic sign when Bernheim's syndrome is suspected in a case with left ventricular enlargement. Glushein and Geer[37] similarly believe that the fluoroscopic findings, as observed in their case, are almost pathognomonic of the syndrome of Bernheim. The combination of a large left ventricle with a large right auricle, the two other chambers being of normal size, is not found, according to these authors, in the ordinary types of heart disease. In our first case, fluoroscopic studies seemed to confirm the clinical impression of Bernheim's syndrome. In Case 2, however, right auricular enlargement could not be established fluoroscopically or by kymographic studies.

Case one provided an opportunity of studying the syndrome of Bernheim from the presymptomatic stage to death, a period of three years. Our observations are, for the most part, in accord with the description of the clinical picture recorded by Mazzei.[11] In the first or anatomic period, there are few or no important clinical signs. Interference with the filling of the right ventricle is counterbalanced by dilatation of the infundibular portion of the chamber and by enlargement of the right auricle. There may be some distention of the cervical veins and the venous pressure may be found elevated while the circulation time remains at the upper limit of normal. The second or clinical period, is divided into two stages. In the first stage, there is "dissociated" failure of the circulation; that is, systemic venous engorgement without disturbance in pulmonary blood flow. The lung bases are clear, and dyspnea is absent or minimal while hepatic enlargement, ascites, and dependent edema may be marked. During this interval, the circulation time may still be within upper normal limits or slightly increased while there is appreciable elevation in venous pressure. Fluoroscopic and kymographic studies may establish enlargement of the left ventricle and right auricle with normal size of the two other chambers. It is this stage of "isolated right heart failure" which is recognizable clinically as Bernheim's syndrome. In the second stage, which represents total failure of the heart, disturbance of the lesser circulation is added to the earlier symptoms of venous obstruction. It is at this time that dyspnea and orthopnea become evident. Regarding pathogenesis, however, our findings support the view of Martini and Joselevich[17] that the terminal appearance of pulmonary congestion indicates failure of the left ventricle, and not the right ventricle as supposed by Mazzei.

From cases thus far reported in the literature, it appears that Bernheim's syndrome may develop in any condition capable of producing marked left ventricular hypertrophy and dilatation such as hypertension, arteriosclerosis, chronic nephritis, and aortic and mitral valvular disease.

SUMMARY AND CONCLUSIONS

1. The syndrome of Bernheim is a distinct clinical entity characterized by systemic venous engorgement without pulmonary congestion.

2. This picture of "isolated right heart failure" is the result of stenosis of the cavity of the right ventricle through displacement of the interventricular septum due to marked enlargement of the left ventricle.

3. The diagnosis is suggested when a patient with left ventricular hypertrophy shows signs of right heart failure as the first indication of circulatory embarrassment.

4. The "right heart failure" is not due to myocardial weakness but results from obstruction to blood flow through the right ventricle.

5. Dyspnea and other signs of pulmonary congestion are conspicuously absent or minimal until the terminal stage of the disease when failure of the left ventricle finally supervenes.

6. In a typical case, the circulation time may remain within normal limits for some time while the venous pressure is appreciably elevated.

7. Fluoroscopy may be helpful in demonstrating enlargement of the left ventricle and right auricle with normal size of the other two chambers.

8. The bulging and thickened interventricular septum may influence the electrical axis of the heart and even be responsible for unexpected right axis deviation in cases of left ventricular hypertrophy.

9. The syndrome of Bernheim may occur in any condition causing marked left ventricular hypertrophy and dilatation.

10. Three cases of this syndrome are presented in two of which the diagnosis was made during life and confirmed at necropsy while, in the third, the condition was discovered unexpectedly at post-mortem examination.

The authors wish to express their thanks to Dr. William H. Gordon for some of the essential data in Case 1 and to Dr. Lawrence Sophian, who performed the post-mortem examination.

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PERIPHERAL VASCULAR CHANGES IN DERMATOMYOSITIS

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IN THE differential diagnosis of occlusive diseases of the arterioles, one does not ordinarily think of so-called dermatomyositis. In the nomenclature[1] published by the Nomenclature Committee of the Section for the Study of the Peripheral Circulation of the American Heart Association, no mention is made of dermatomyositis, although such diseases as periarteritis nodosa and lupus erythematosus disseminatus are listed. Herrmann[2] in a recently revised textbook on diseases of the heart and arteries omitted dermatomyositis entirely. In a lengthy review[3] of this textbook, appearing in the *Archives of Internal Medicine,* the author is specifically taken to task for this omission.

There may be several reasons for the omission of dermatomyositis in the classification of vascular diseases. First, dermatomyositis is a rare disease and frequently goes unrecognized.[4, 5] Secondly, there seems to be a lack of unanimity as to just what dermatomyositis represents. Clinically, dermatomyositis, scleroderma, disseminated lupus erythematosus, the Libman-Sacks syndrome, and polyarteritis nodosa appear at times related, and the question has been raised whether or not there is a common denominator in all these

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vaguely understood diseases.[7, 8] Finally, the term dermatomyositis is misleading. In standard textbooks of medicine[6a, b] the disease may be found listed under such a heading as nonsuppurative myositis. Theoretically, the term dermatomyositis is applicable to any disorder in which there is inflammation of the muscles associated with skin changes. O'Leary and Waisman[4] point out, however, that cutaneous manifestations may be totally absent. It appears that the cutaneous and muscular features of dermatomyositis have been unduly emphasized. In certain patients, the peripheral vascular changes dominate the entire clinical picture. Jager and Grossman[5] observed a history of Raynaud's phenomena in four out of their nine reported cases of dermatomyositis. The following case report illustrates the pronounced peripheral vascular features of this illness.

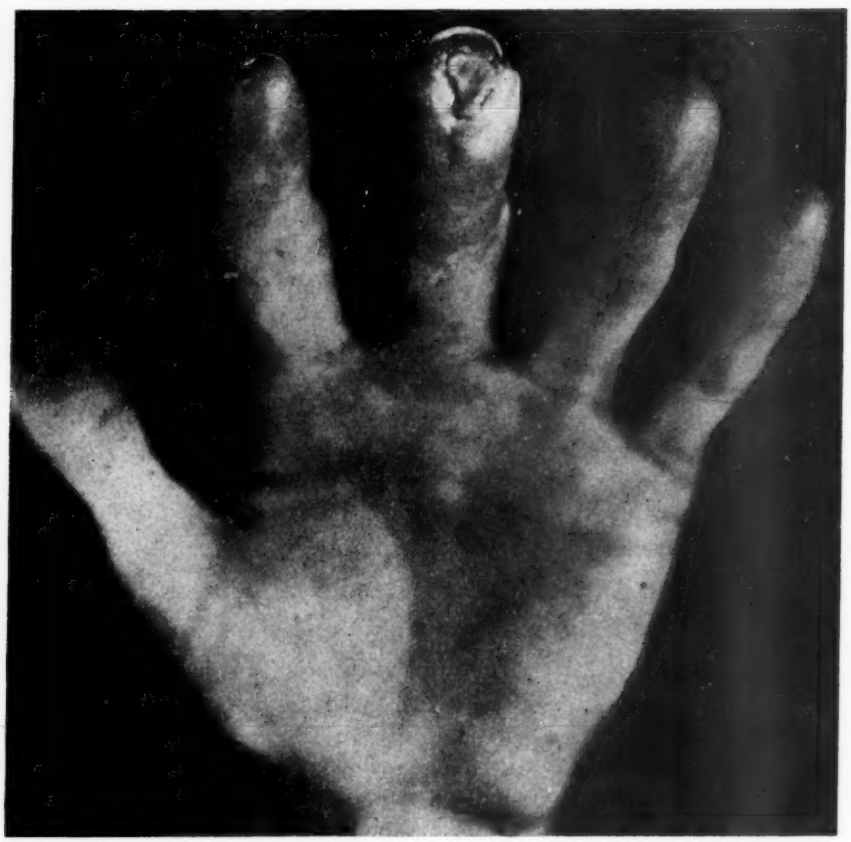

Fig. 1.—Photograph showing trophic ulcer of left middle finger.

CASE REPORT

L. J., a 35-year-old, white, Jewish soldier, was in good health until May, 1943, when he developed a vesicular and papular type of eruption of his hands and feet. At the time he was stationed in a Southwest Pacific Area and was cared for by the staff of a station hospital unit. He was told he had a fungus infection and various local applications, including sulfonamides, were tried with periodic relief. About the time of the onset of his skin condition, he gradually developed weakness, abdominal pain, vague aches and pains in most of his muscles, cramps in his legs on walking and "nervousness." His appetite was poor, and he lost 25 pounds in weight. In March, 1944, the condition of his skin was much improved, but he developed an ulcer at the tip of the middle finger of his left hand. This ulcer was refractory to treatment. He was finally evacuated to the United States with a diagnosis of dermatitis of unknown origin and psychoneurosis.

Physical Examination.—The patient was well developed but undernourished and appeared chronically ill. The skin over the distal portion of his lower extremities had a slight gloss and seemed slightly adherent to the underlying structures. The palms and soles showed a few vesicles and some scaling, with evidence of atrophy. The left middle finger, at its distal volar aspect, showed an indolent-appearing ulcer. The ulcer had a necrotic slough over its base. There was tenderness over most of the large muscles, including the deltoid, trapezius, and triceps. Both hands and feet felt unusually cold, and in the dependent position there was marked rubor. The dorsalis pedis pulsations were not elicited, but the posterior tibial, popliteal, and radial pulsations were easily palpated. The left radial pulsation felt weaker than the right. Elevation of the lower extremities for a period of three minutes caused the feet to take on a blanched, cadaverous appearance. The heart and lungs were normal; the blood pressure measured 116/80; the spleen was not palpable, but there was moderate generalized lymphadenopathy. The reflexes were normal, but slightly hyperactive. Examination of the peripheral nerves by electrical stimulation showed spotty and irregular changes of both sides of the body. These changes did not appear to be organized in any special nerve distribution but occurred rather in a pattern referable to terminal motor branches. An oscillometric study revealed no pulsation over the dorsum of each foot and relatively less of a pulsation in the left wrist (Table I).

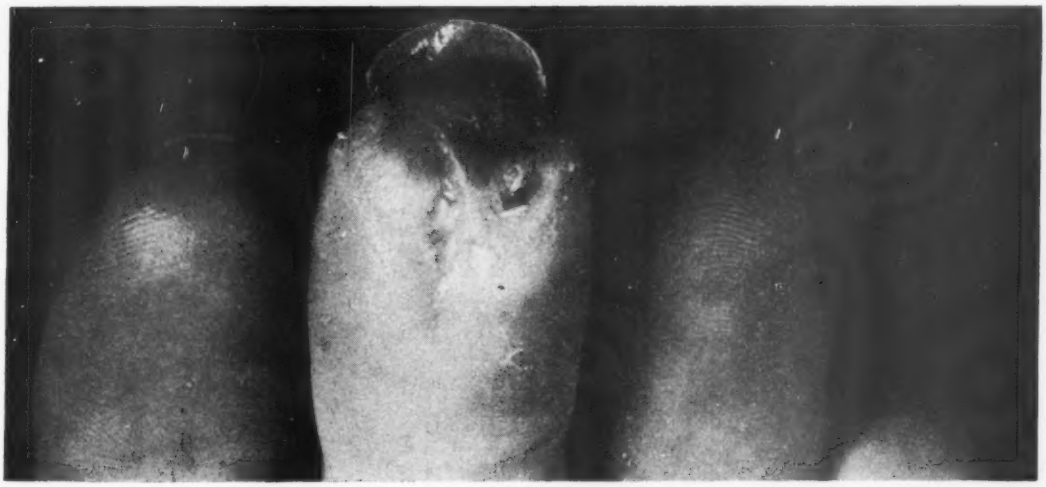

Fig. 2.—Photograph showing same indolent ulcer after six weeks of conservative treatment.

Laboratory Studies.—There was no anemia. The leucocyte count varied from 12,000 to 17,000 per cu.mm. The differential count was normal except that the monocytes occurred in a proportion of from 6 per cent to 12 per cent. The sedimentation rate was 15 mm. in one hour (modified Cutler method used; normal less than 10); the urinalysis was negative; the blood Kahn test was negative; blood smears for malaria were negative; a stool examination for parasites was negative; the blood cholesterol was 270 mg. per 100 c.c.; creatinine, 1.37 mg. per 100 c.c.; blood calcium, 10.4 mg. per 100 c.c.; total serum protein, 6.6 Gm.; globulin, 2.4 Gm. and albumin, 4.2; albumin-globulin ratio, 1.7; urinary creatine and creatinine studies revealed a creatine value of 112 to 51 mg. in 24 hours and a creatinine value varying from 1.56 to 1.52 Gm.; basal metabolism, -1 per cent; skin test for trichiniasis, negative; electrocardiograms, negative; x-ray of heart and lungs, normal; x-ray of left middle finger showed early atrophy of bone in the distal phalanx. X-ray examination of both feet was negative. No calcification was seen.

TABLE I. OSCILLOMETRIC STUDY

	RIGHT	LEFT
Calf	5.00	5.00
Ankle	1.80	1.90
Foot	0.00	0.00
Wrist	1.90	1.50

Biopsy.—A biopsy of the lymph node taken from the left epitrochlear region, on the same side in which the ulcer of the finger was located, showed evidence of inflammation. Several of the vessels in the surrounding tissue showed almost complete occlusion. Biopsies of the left soleus and the left deltoid muscles showed an increase in the nuclei of the sarcolemma. Here and there the bundles were covered with a few round cells, an occasional neutrophile, and a phagocyte. The interstitial stroma was infiltrated with plasma cells, small round cells, and occasional neutrophiles and phagocytes. The capillaries were surrounded by small round cells and plasma cells, and diapedesis was present. The walls of the medium sized vessels occasionally were infiltrated with round cells. There was no evidence of endothelial proliferation. The pathologist concluded: "There is a myositis with some muscular degeneration and atrophy, and there is a vascular disturbance, especially noted in the capillaries and smaller vessels" (Figs. 4, 5, and 6).

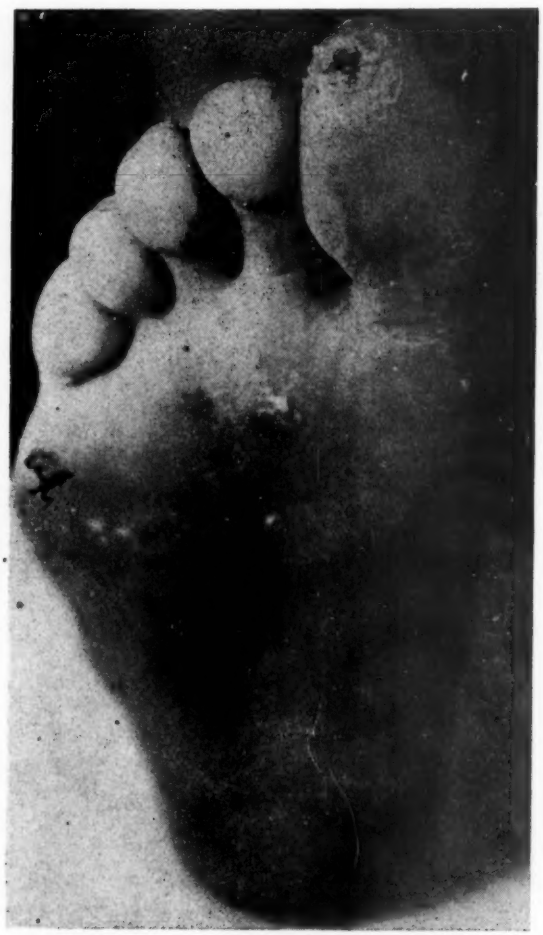

Fig. 3.—Photograph showing beginning trophic changes of the large right toe.

Course.—The patient was observed for approximately two months at this general hospital, during which time his chief complaints were generalized pains and aches, particularly in the lower extremities after walking, a nonhealing ulceration of the left middle finger, and progressive weakness. Writing a simple letter was enough to tire him. He was afebrile except for three episodes of fever lasting less than two days. Each bout of fever reached 100.5° F. Prior to hospitalization he was a heavy smoker, averaging two packages of cigarettes a day. This habit was discontinued after much persuasion. The ulcer on the finger was followed by the surgical service. Local applications of a bland ointment, together with physiotherapy, were prescribed. In the last five weeks the ulcer showed a tendency toward healing. Modified Buerger's exercises were given daily, but there was no appreciable improvement in the circulation of his lower extremities. A paravertebral block on the left side was performed. The

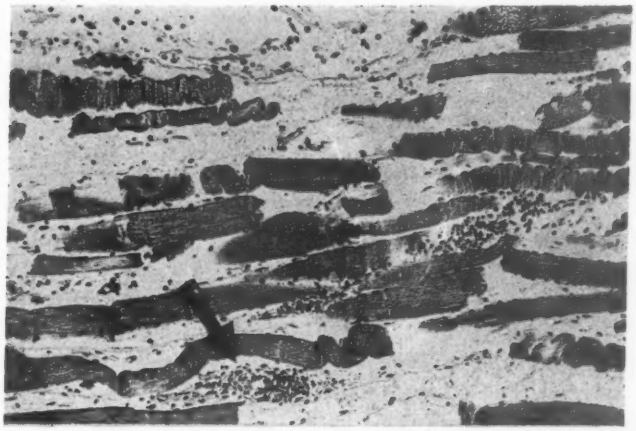

Fig. 4.—Photomicrograph of low-power magnification illustrating muscular degeneration. Note interstitial infiltration with plasma cells and phagocytes. Arrow indicates perivascular cuffing. Section from soleus muscle.

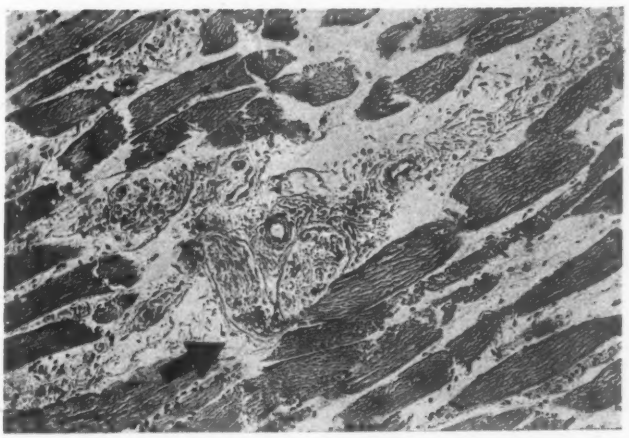

Fig. 5.—Photomicrograph of low-power magnification of a section from the deltoid muscle. Arrow points to small round-cell infiltration in muscle bundles.

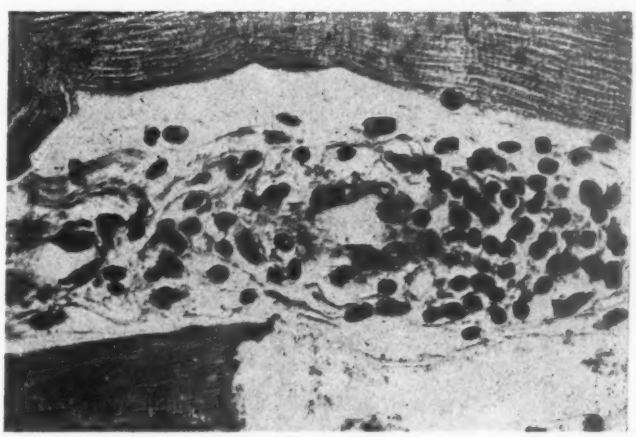

Fig. 6.—Photomicrograph of high-power magnification illustrating the small round cell perivascular infiltration.

left lower extremity became warmer but there was no increase in the oscillometric readings. A brachial block was also tried on the left side. The left hand became warmer and there was definite relief of discomfort in the ulcerated finger. He was seen by the ophthalmologist who found narrowing of all the retinal arteries. The patient, however, had no complaints referable to his eyes.

DISCUSSION

Clinically, the peripheral vascular manifestations in this patent were similar to those seen in thromboangiitis obliterans. He was in the relatively young age group, of Jewish blood, and smoked excessively. The pathologic findings, however, revealed none of the endothelial changes characteristic of this disease. Periarteritis nodosa is suggested by the mild leucocytosis, the occasional bouts of fever, and the widespread and varied distribution of the clinical findings. Again, the pathologic findings were not characteristic of the usual case of periarteritis nodosa. The changes in the walls of the blood vessels seemed more in the nature of an infiltration without the necrosis seen in periarteritis. A skin biopsy taken from the region where the epitrochlear gland was removed showed no significant findings. Specimens taken from widely separated muscles, however, showed unequivocal evidence of a myositis such as has been reported in dermatomyositis (Fig. 4). Of particular interest are the pathologic changes seen in the arterioles (Figs. 5 and 6). The concept that dermatomyositis may be primarily a disturbance of the blood vessels was first proposed by Lépine,[9] in 1901. The term "angiomyositis" was suggested. Certainly, the clinical course and manifestations of our patient lend support to the concept that dermatomyositis is primarily a vascular disease.

SUMMARY

1. A patient with certain features of so-called dermatomyositis is presented who showed pronounced peripheral vascular disturbances.

2. The widespread vascular changes in dermatomyositis are emphasized. In the differential diagnosis of occlusive diseases of the arterioles, so-called dermatomyositis should be included.

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THE RECRUIT'S HEART

Reduplication of the First Sound: Heart Strain and a New Method of Calibration

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I PUBLISHED a preliminary report on this subject in 1940, and I have since made a systematic examination of a further number of hearts in an endeavour to learn the significance of reduplication of heart sounds and the effect of strain on the heart. I have again, largely, made use of the hearts of young people, for they react more quickly and delicately to sudden strain. This has proved to be a fertile field which may stimulate and encourage other workers to improve on my results.

It is not my intention to deal with the bibliography of the subject of added heart sounds. I would refer those who are interested to a recent valuable paper by Evans, "Triple Heart Rhythm."[2] Moreover, I have to a certain extent followed Evans' schematic pattern when illustrating the cardiac cycle (Fig. 1).

I first drew attention in 1919 to the fact that reduplication was much more common in young children than in adults. Cossio and Braun-Menendez[4] (1935) gave the following percentages: infants, 12 per cent; adolescents, 7 per cent; and adults, 1 per cent.

Many are the theories, which have been advanced in an effort to explain the mechanism producing added sounds, and Pillsbury[5] points out that they are as variable as possible. Whilst most of the explanations have dealt with asynchronism of the closure of the aortic and pulmonary valves, Obrastzow[6] (1905) states it indicates the beginning of cardiac degeneration, and Core[7] (1912), dealing with mitral stenosis, suggests that audibility of the opening of the mitral valves is an explanation of double second sounds. Lian[8] (1934) says that reduplication of the second sound and the third heart sound are two distinct phenomena; White[9] (1939) associates accentuation of the third heart sound with exercise and dilatation of the left ventricle, whilst Sabathie[10] (1938) refers to respiration, hypertension, and ventricular asynchronism as being important factors. One of the objects of this paper is to try to prove that reduplication *after exercise* is never of the second sound, but always of the first sound, and that it is due to asynchronism of ventricular contractions brought on by dilatation of the right ventricle.

RELATIONSHIP OF YOUTH AND REDUPLICATED SOUND

I (1940) have described elsewhere the systematic examination of the hearts of young subjects in the presence of other doctors, who can confirm the results. Briefly, the subjects consisted of a group of seventeen children between the ages of 6 and 8 years, a group of nine children between the ages

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of 8 and 10 years, and a group of twenty children between the ages of 10 and 12 years. Subsequently a group of R.A.F. recruits were examined at Halton; nine were nonathletic, untrained recruits and twenty-two had undergone mild training. Lastly, a group of twenty-one Home Guards were examined.

On analysis I found that 72 per cent of children between the ages of 6 and 12 years have reduplication of sounds either at rest or after exercise. In boy cadets between the ages of 14 and 20 years, 29 per cent had reduplication either at rest or after exercise. In R.A.F. recruits between the ages of 17 and 39 years, who had only mild training, the percentage had dropped to 19 per cent. Finally, among the Home Guards, after the age of 40 years, the percentage had dropped to nil.

I am of the opinion that, provided the exercise be severe enough, a change of rhythm can be induced in the hearts of most children under the age of 10 years.

Reduplication of First Sound After Exercise.—As already mentioned the hearts were examined in the presence of other doctors. I was puzzled by the fact that they were unable to hear the reduplication and to confirm my findings, until I noticed that they listened at the base, whereas I unconsciously listened over the apex. I afterwards found that I myself was unable to pick up the reduplication at the base.

Systole	Diastole	Systole	Diastole	
LUP	DUP	LUP	DUP	Normal rhythm at rest
LUP 1	DUP lup 2 r	LUP 1	DUP lup 2 r	Group 1 after exercise
lup LUP r 1	DUP 2	lup LUP r 1	DUP 2	Group 2 after exercise

Fig. 1.—Diagram showing position of reduplication of first sound in cycle in the two groups after exercise. *r*, reduplicated sound.

Let us consider the heart sounds from an onomatopoeic standpoint (Lup being the first sound and Dup being the second sound). On lying down, immediately after exercise, not only is a reduplication noticeable, but more often than not, a systolic "whiff" can be heard synchronizing with the Lup, or first sound. The other two sounds will therefore be the true second sound and the reduplicated sound (but not necessarily in that order). Having identified these sounds, if the stethoscope be then slowly slid, without lifting, from apex to base, it will be found that the reduplicated sound becomes more and more indistinct and disappears as the base is approached. The true second sound, on the other hand, becomes more and more audible and distinct towards the base. Although I and others have searched systematically on innumerable occasions, we have never yet been able to hear two distinct Dups at the base, as a result of exercise in these young hearts. I have come to the conclusion *that what I had previously deemed to be a reduplicated second sound, is in fact reduplication of the first sound,* and that it is a ventricular phenomenon, and has nothing to do with the second sound.

In certain boxers who have broken down in training, it has been found, after a period of rest, that the rhythm has changed from Group 1 to Group 2, only to change back again after resumption of training.

In short the sounds after exercise are Lup Dup lup or lup Lup Dup never Lup Dup dup. Tables I, II, and III should suffice for purposes of comparing and contrasting the old and the young.

These and other tables show that reduplication can easily be induced in young people; that the younger the subject the more easily it can be induced; that it can be brought on by exercise; that it becomes more noticeable on lying, but, on the other hand, when the subject is in the knee-elbow or prone position, or in the erect position, it becomes more indistinct. The reduplication should be sought for most diligently and carefully during the first few beats after exercise, for like the presystolic murmur of early mitral stenosis it may be evanescent and elusive.

Dilatation of the Heart After Exercise.—Thirty teleroentgenograms of the hearts of children were taken immediately before and after exercise. These were traced and charted. The charts were measured by means of an instrument

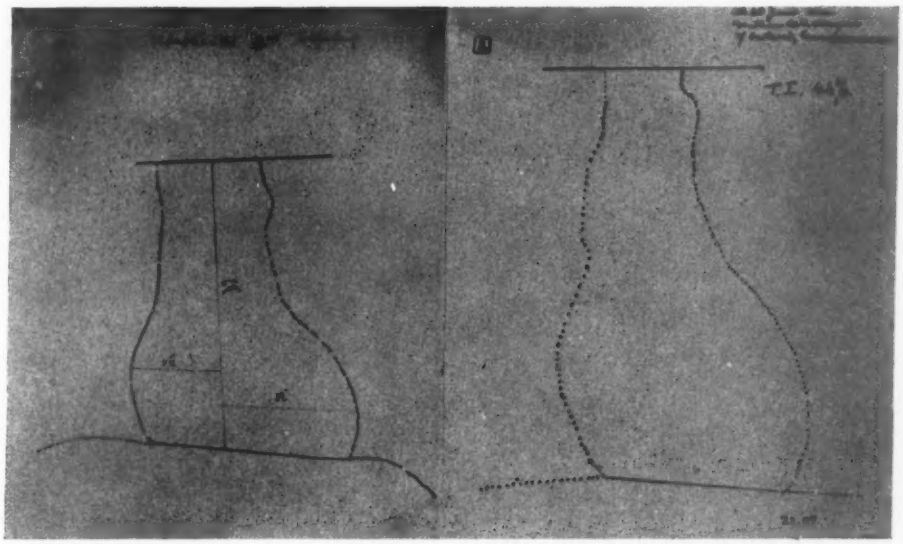

Fig. 2. Fig. 3.

Fig. 2.—Chart of Public School boy boxer, aged 16 years, heavy weight. Area: 23.08 square inches.

Fig. 3.—Chart of Canadian champion, aged 20 years, featherweight. Area: 22.87 square inches.

TABLE I

AGE (YRS.)	REDUPLICATION ON LYING	REDUPLICATION ON LYING AFTER EXERCISE	REDUPLICATION STANDING AND PRONE POSITION	DURATION AFTER EXERCISE
7	0	0	0	0
7	0	+	0	Disappeared in 30 seconds
6	+	+	+	Persisted indefinitely in recumbent position
7	+	+	0	Persisted indefinitely in recumbent position
7	0	+	0	Disappeared in 30 seconds
7	0	+	0	Disappeared in 20 seconds
7	0	+	0	Disappeared in 30 seconds
7	0	+	0	Disappeared in 30 seconds
9	0	+	0	Present for first few beats only
9	Waxing and waning with respiration	+	0	Waxing and waning with respiration indefinitely

known as a *planimeter*, and the sectional areas in square inches were calculated down to two decimal places. The increase in the sectional area after exercise was found to be 8.75 per cent. (The subjects each did fifty skips.) Other interesting figures were obtained, as for example the heart of a child increases by about 1 square inch per year (some of these children have been x-rayed at yearly intervals). The area of the heart of a child of 10 years is, on the average, about 12 square inches, whereas the area of the heart of a boxer aged 22 years averages about 22 square inches. The heart of Kilrain, an old champion, aged 75 years, who fought Fitzsimmons, was 28.07 inches (Fig. 5).

That the right side of the heart is the first to dilate after exercise can be shown as follows. Each diagram was divided by a median vertical line running down the vertebral spines (Figs. 2, 3, 4, and 5). In this way a constant division of the heart into right and left sides was obtained, and it was found that the areas of the right sides of these hearts after exercise had increased by

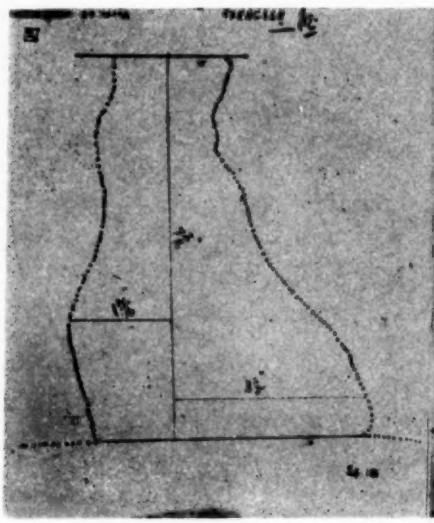

Fig. 4.

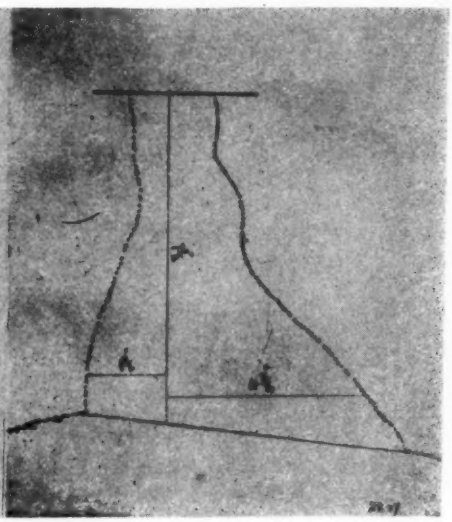

Fig. 5.

Fig. 4.—Chart of English champion, aged 35 years, middle weight. Area: 24.10 square inches.

Fig. 5.—Chart of Australian champion, aged 75 years, heavy weight. Area: 28.07 square inches. Note how pattern changes to left in the last subject.

TABLE II

AGE (YRS.)	REDUPLICATION ON LYING	REDUPLICATION ON LYING AFTER EXERCISE	REDUPLICATION STANDING AND PRONE POSITION	DURATION AFTER EXERCISE
10	0	+	0	Occasional after deep respiration
10	0	0	0	0
12	0	0	0	0
11	+	+	0	Persisted indefinitely in recumbent position
10	+	+	0	Persisted indefinitely in recumbent position
11	0	0	0	0
10	+	+	0	Persisted indefinitely in recumbent position
12	0	0	0	0
11	0	0	0	0
11	+	+	0	Persisted indefinitely in recumbent position

14.30 per cent. When this figure is compared with the percentage increase of the heart as a whole, 8.75 per cent, it seems probable that dilatation of these young hearts, after exercise, is largely, if not entirely, due to right-sided dilatation. This is in agreement with Roy and Adami's concept of the heart and its walls as those of a sphere or spheroid, and the law of strain upon the walls of a spheroid. Just as the thinnest part of a football bladder or tire is the first to bulge on increase of pressure, so the right side of the heart is the first to dilate as a result of intracardiac pressure, for the walls of the left ventricle are three times the thickness of the right ventricle. This dilatation of the right ventricle is the reason reduplication is brought on by exercise in these young hearts, and, furthermore, it is also my opinion that the association of reduplication (triple rhythm) with mitral stenosis is due to the same cause, namely dilatation of the right side of the heart. In short, reduplication of the first sound is always an indication of the dilatation of the right side of the heart whether it be in disease or after exercise.

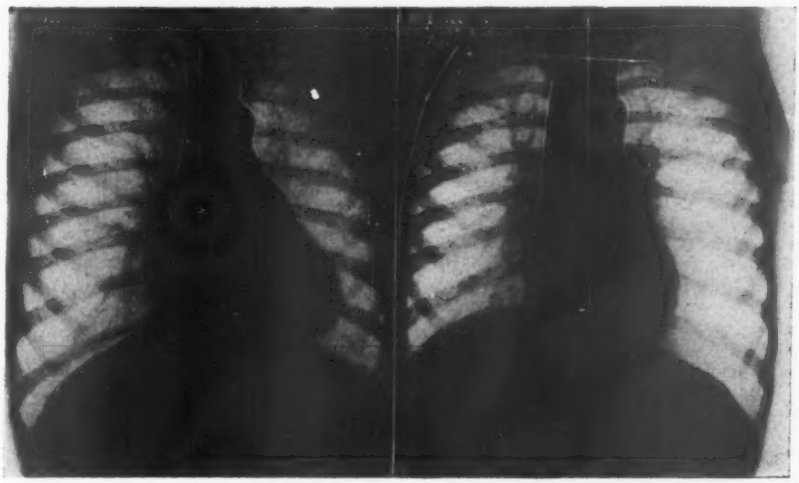

Fig. 6 Fig. 7.

Fig. 6.—Lying on left side, heart moves to the left.
Fig. 7.—Lying on right side, heart moves to the right.

The Effect of Posture on the Position of the Heart in the Thorax.—By means of a platform, subjects were x-rayed in various postures: lying on the left side, lying on the right side, suspended head downwards, and in erect, prone, and supine positions. The heart's position was found to vary with posture, e.g., when lying on the left side the heart falls towards the left, when lying on the

TABLE III. HOME GUARD

AGE (YRS.)	REDUPLICATION ON LYING	REDUPLICATION ON LYING AFTER EXERCISE	REDUPLICATION STANDING AND PRONE POSITION	DURATION AFTER EXERCISE
47	0	0	0	0
42	0	0	0	0
43	0	0	0	0
48	0	0	0	0
47	0	0	0	0
42	0	0	0	0
54	0	0	0	0
48	0	0	0	0
46	0	0	0	0
49	0	0	0	0

right side, the heart moves to the right, when standing on the head it moves downwards, dragging on the diaphragm (Figs. 6, 7, 8, and 9).

I hope later to demonstrate not only the effects of gravity on the position of the heart in the thorax and the way this varies with different postures, but, also to show that, to some extent, it rotates on its axis; this is quite feasible, since the heart is comparatively loosely suspended in the thorax and pericardium, and even the fibrous pericardium is largely interspersed with elastic fibers.

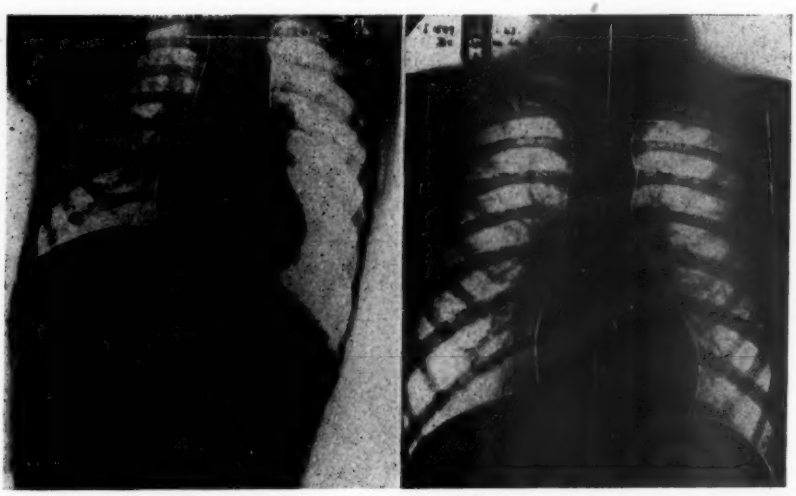

Fig. 8. Fig. 9.

Fig. 8.—Standing on head, heart moves downward, dragging on the diaphragm.
Fig. 9.—Normal erect position.

The Audibility of Murmurs.—The audibility of a murmur, reduplication, or other cardiac sound varies with the proximity of the chest piece of the stethoscope to the source of origin of such murmur or reduplication. For example, the presystolic murmur of mitral stenosis is more audible on lying on the left side than when lying on the right, because the chest piece is nearer to the left side of the heart. The diastolic murmur of aortic regurgitation is more easily picked up in the prone position, or when the patient leans forward, because gravity causes the heart to more nearly approach the chest piece of the stethoscope. The approximation of the chest wall to the heart is the reason a systolic murmur is loudest at the end of expiration, and the presumption that, if a murmur disappears during the inspiratory phase, it must be innocent, is unsafe.

Diastolic Murmurs.—Not only have I had the opportunity of investigating diastolic murmurs in children suspended head downward, but I have also been fortunate enough to study the murmur in a gymnast suffering from double aortic disease. He could balance himself on his head for an indefinite period. My previously conceived ideas on the subject have changed. I had in the past assumed that the diastolic murmur of aortic regurgitation was influenced by gravity, but, on examining the subjects suspended head downwards, it was found that there was no difference in the audibility of the murmurs as compared with the erect position. I have come to the conclusion that the mechanism of the aortic diastolic murmur is that of backward suction; hence, it is well heard along the right border of the sternum as low as the cardiac impulse.

Diastolic murmurs are higher in pitch than systolic murmurs. Furthermore, the murmurs can be artificially produced by squeezing the bulb of a syringe; it will be found that the same rule applies—the murmur of relaxation is higher pitched than that which accompanies the squeeze. A point worthy of emphasis is that the subject with aortic disease could stand on his head for a much longer period than subjects who were used as controls; the latter soon became distressed complaining of buzzing in the ears and fullness in the head, whereas the former could remain balanced for a long period without apparent discomfort.

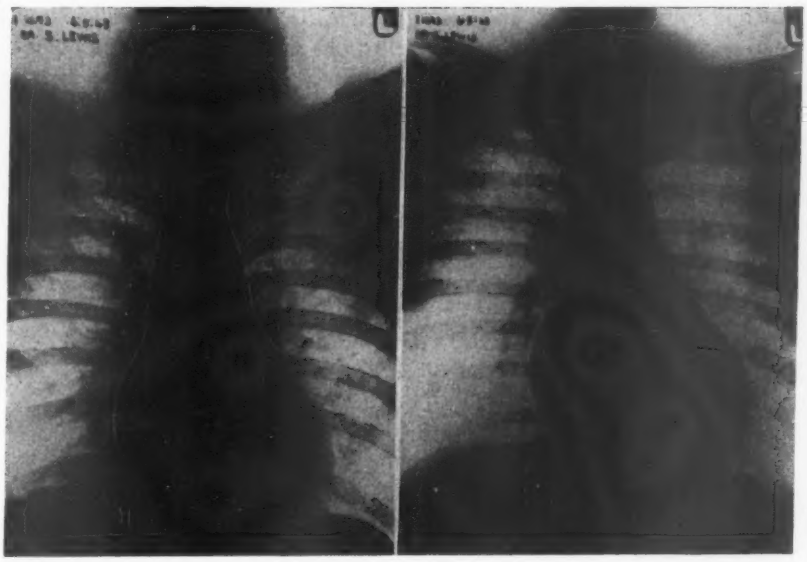

Fig. 10. Fig. 11.

Fig. 10.—Prone. Area: 31.40 square inches.

Fig. 11.—Supine. Area: 33.80 square inches. Note how the median vertebral line shows that the heart has rotated on its axis, and how the sectional area has changed from 31.40 square inches to 33.80 square inches. The area of this particular heart in the erect position was 27.95 square inches.

Why is the reduplication more audible on lying? In the upright position the right ventricle lies between the sternum and the left ventricle, and only a very narrow strip of the left ventricle is concerned with the anterior surface of the heart. In the supine position, especially after dilatation, the effects of gravity make this relationship still more slender, and moreover this can be demonstrated by teleroentgenograms taken in the prone and supine positions, for, not only do the sectional areas of the hearts vary with change of positions, but the median vertical line shows that the heart, as a result of gravity, rotates on its axis. In the supine position the heavy left side sinks still further from the chest wall into the thorax, its place being taken by the lighter right side, which comes nearer the chest wall. This is even more marked when the right ventricle is dilated as a result of exercise in these young hearts (Figs. 10 and 11).

In other words in the supine position the right ventricle more nearly approaches the chest piece of the stethoscope than in the prone or knee-elbow position, and this explains why reduplication can be so easily picked up in the supine position as compared with the knee-elbow or prone position. This follows the law of floating spheres, i.e., when unevenly weighted, the lightest

part comes to the surface. (It is well known that changes in the electrical axis occur with change in body position.)

Heart Strain.—The influence of sudden, violent, or unduly prolonged exertion has been well described by Allbutt.[12] During the last twenty years or so it has become the fashion to assume that the healthy heart cannot be harmed by mere physical strain. I am credited with having examined more hearts of various athletes (swimmers, cyclists, weight-lifters, boxers, football players, runners, etc.) than anyone else in this country, and I have given elsewhere examples of hearts of champion athletes which have been damaged by strain and strain alone; no other cause could be found. If the heart of a champion breaks down, who is there to prove whether or not that heart was healthy at the commencement of his athletic career? At any rate, these hearts were healthy enough to enable these particular athletes to attain championship class. The only perfect heart that I can envisage is one kept beating in a glass case, free from the vitiation and vicissitudes of life.

For convenience let us divide heart strain into (a) mild heart strain, (b) moderate heart strain, and (c) severe heart strain.

TABLE IV. BOXERS' HEARTS; RATE OF PULSE BEFORE AND AFTER COMING OUT OF RING
(March 22, 1943)

	PULSE BEFORE ENTERING RING	PULSE AFTER 3 ROUNDS ON REACHING DRESSING ROOM	PULSE 5 MINUTES LATER
1	84	180	120
2	92	190	160
3	84	146	120
4	120	184	160
5	92	128	88
6	100	196	166
7	104	192	140
8	92	192	128
9	96	168	80
10	104	140	120
11	88	112	110
12	68	140	90
13	88	112	160
14*	88		100
15	76	140	110
16	120	176	140

*Knocked out after second round. Incidentally, immediately after the knock out, the pupils and knee jerks were found to be unequal.

a. *Minimum Heart Strain:* As has already been shown, a mild strain of only fifty skips increases the sectional areas of the heart by 8.75 per cent. In very young hearts dilatation is even brought about by taking a few deep breaths or by mere change from the upright to the recumbent posture.

b. *Moderate Heart Strain:* The pulse rates and blood pressures of a number of service boxers taking part in three two-minute rounds were taken, and the results can be seen in Tables IV and V. There was a vast increase in both the pulse rates and the blood pressures at the end of the contests. On the other hand there was a slight decrease in the average diastolic pressure on reaching the dressing room, and indeed I have found that the diastolic pressures of champion weight-lifters are lower after the "lift" than at rest. It will be realized that there is an enormous strain on the heart even after only three two-minute rounds and that the effect on some hearts is greater than on others (Tables IV and V).

c. *Severe Heart Strain:* The following case* demonstrates the effect of severe heart strain.

*The notes of this case were kindly given to me by Dr. Ewart Evans of Birmingham.

TABLE V. BOXERS'* BLOOD PRESSURES; THE BLOOD PRESSURES BEFORE ENTERING THE RING AND IMMEDIATELY ON REACHING DRESSING ROOM AFTER FIGHTING THREE TWO-MINUTE ROUNDS
(March 28, 1943)

	BEFORE ENTERING RING		AFTER REACHING DRESSING ROOM	
	SYSTOLIC	DIASTOLIC	SYSTOLIC	DIASTOLIC
1	145	85	180	80
2	140	82	188	70
3	145	82	180	80
4	128	80	150	60
5	140	80	150	75
6	160	60	not taken	
7	110	80	180	70
8	152	82	145	78
9	145	82	190	90
10	150	80	140	70
11	128	80	140	70
12	158	88	165	80
13	130	80	140	70
14	160	80	165	80
15	160	82	180	80

Before entering the ring the average systolic pressure was 143, and the average diastolic pressure was 79.

After reaching dressing room the average systolic pressure was 164, and the average diastolic pressure was 75.

It has also been found that the diastolic pressures of champion weight-lifters are lower after the "lift" than at rest.

*Average age, 22 years.

"T. W. (male) aged 27 years, solicitor, very healthy, stockily built type. No previous history of any illness. Played a very strenuous and hard fought game of 'Rugby Football.' Position—stand off half. A few minutes after the termination of the game he died.

"*Autopsy, the same evening—showed dilatation of right heart, left ventricle in a state of spasm.* I know that his heart was of normal size, and state, and that there were no murmurs or any signs suggesting any cardiac lesion before the game."

This case is noteworthy, and should be historic, for seldom, if ever, can there have been so little time lost in performing a post-mortem examination. Not only was dilatation of the right heart discovered, but also the left ventricle was still in a state of spasm. The post-mortem findings bear a striking resemblance to those found in thiamine deficient dogs: right auricle and ventricle dilated and the left ventricle in apparent contraction.[14] It is also one more link in the chain of evidence that it is the right side which first bears the brunt after strain.

I can recall two further instances of severe heart strain which have come under my personal care; they are worthy of comment if not of emphasis. A French marathon runner, after finishing the course from Windsor to London, had complete heart block for twelve hours. I also examined Hood on the night he died at the National Sporting Club; after winning every round up to the seventeenth, he fell down unconscious, not from a blow, and never recovered.

I have elsewhere referred more fully to these and other examples of the effect of severe strain on the heart.

Training.—Cardiac endurance is measured by the ability of a heart to resist dilatation, and its ability to regain its former volume after dilatation. Gradual training brings this about by gradually increasing the tonus of the heart muscle. Roy and Adami's[11] law states: "the strain upon the walls of a sphere or spheroid increases with its circumference, and therefore, the resistance to contraction of the heart wall is increased whenever it becomes dilated"; i.e., the more a heart is dilated the more difficult it is for it to regain its former volume. The danger of sudden muscular effort is to produce transitory dilata-

tion of the heart and when these sudden muscular efforts become frequent, a permanent dilatation ensues, as Lord Horder,[15] in a personal communication, pithily suggests, "repeated ventricular stretching must spell *harm* eventually." If the process of training be gradual and successful, the heart, instead of becoming permanently dilated, will assume a physiologic hypertrophy. It will be recalled that reduplication is uncommon as a result of exercise in the hearts of those over the age of 35 years. The heart after that age loses its dilatability and elasticity, which explains why it is difficult for a man above this age to recover second wind. The heart is like an engine, acceleration and deceleration should be gradual. It was noticeable that Lovelock and other great athletes, at the finish of a grueling race, continued for another lap, gradually decelerating; certain greyhounds, e.g., Mick the Miller, instinctively acquired the same knack. An athlete should not retire from games suddenly. Professional golfers live longer as a class than professional football players and boxers, for too many of the latter give up exercise suddenly in favor of sedentary occupations such as hotelkeeping.

Stitch in Side and Second Wind.—A certain number of recruits invariably fall out, during the course of a match, complaining of pain in the side, sometimes one side, sometimes the other. In my experience the most common location of pain is in the left hypochondrium.

I have devoted a considerable amount of time to the study of stitch and second wind, questioning numerous and various athletes, runners, rowers, boxers, swimmers, etc., and I have come to the conclusion that there are two kinds of "stitch": (1) superficial, and (2) deep.

The former has been well described by Capps,[17] in 1941, and in the main I cannot cavil with his theory that it is due to anoxemia or ischemia of the diaphragmatic muscle. I would like to add that spitting has been found by generations of schoolboys to be a useful factor for giving relief. It was an added ritual that one must lift a stone, spit on it, and replace it on the same spot. This was considered to be an infallible panacea, though I have no doubt that the temporary rest and the bending over were the true reasons for the relief of the pain.

The *deep* stitch is also due to ischemia; it is dull in character, a sense of constriction or pressure, constant in position in the precordium. It is akin to cramp or angina. Jenner,[16] in a letter to Heberden, in 1778, first implied the connection between ischemia and angina. I am not unmindful of the fact that the word ischemia was not then invented, yet the condition itself was well known. The explanation at which I have arrived, as a result of a wide and varied experience of all classes of athletes, is as follows: Sudden and undue effort causes the heart to contract vigorously. The metabolism of the heart, when in vigorous action, requires more frequent flushing with blood, than when it is quiescent. The forcible contraction interferes temporarily with the increased needs of the heart. It is a state of relative *ischemia* in which the coronary arteries do not admit enough blood for sudden and increased effort, thus causing pain. The pain is relieved by relaxation of the heart musculature, just as relief of cramp in the calf is brought about by flexing the big toe (which causes the calf muscles to relax).

The relaxing of the heart musculature is immediately followed by diminished resistance, and the increased intracardiac pressure acting against this diminished resistance causes dilatation of the cardiac chambers. The pain gradually disappears and the dilated heart develops a new power which enables it to complete an extraordinary task, free from pain, just when all

seems lost—*this is second wind.* I had always been dimly aware that certain athletes never have a stitch in the side, and never have second wind; this was confirmed by T. the famous varsity rowing coach. After an extensive questionnaire of athletes, I have come to the conclusion that stitch occurs only in those who have been injudiciously trained. The successfully trained athlete's heart is a racing machine from the start.

Danger Signals During Training.—The early part of training is important, for it is at the beginning that harm may be done. It is sudden strain on a sedentary heart that is harmful. The way individuals differ can be seen in the pulses of boxers before and after contests. Therefore I would suggest that the present method of submitting all the members of a unit to the same degree of training is unwise; it would be much better to subdivide them into categories, according to the way they stand training. I will now discuss a few danger signals which have appealed to me personally in my association with athletes and recruits.

As adviser to the British Weight Lifting Olympic Team, I had ample chances to study training in all its phases. In weight-lifters it was found that a constant pulse rate of 100 or over at rest, a reduplication of sound, and a sensation of throbbing or pounding in the head on stooping or lying were signals that the training for that particular athlete was too severe. I also came to the conclusion that no youth under the age of 18 years should go in for championship lifts, and moreover as a result of my examination of I. R., the champion woman weight-lifter after breaking the world's record, I formed the opinion that weight-lifting was quite unsuitable for women athletes. It is worthy of comment that this particular athlete had signs of a dilated heart, a well-marked systolic murmur, triple rhythm at rest, and poor exercise tolerance.

The pulse rate of a well-trained athlete is slower than normal, and in this connection I can recall a fight for the championship of the world between A and B. A had a pulse rate of only 56 half an hour before entering the ring, he was quite calm and relaxed, and after fighting furiously for nearly a round his pulse rate was only 110 (the fight ended unsatisfactorily towards the end of the first round). B on the other hand showed every sign of overtraining (a few months before he had been defeated after a punishing fight in America). He was fidgety and nervous during my examination; his heart was enlarged; a loud systolic murmur and reduplication could be heard at rest. His pulse rate was 98 per minute, and on reaching the dressing room his pulse rate was found to be 150 per minute. Soon afterwards, he was knocked out in his last fight before retiring for good. Yet, years afterwards, I found his pulse rate to be normal, which shows that the heart tends to recover provided the athlete slows up in time.

I fully realize that boxers are keyed up just before a fight to such an extent that one would not dare risk taking their blood pressures just before a championship fight, for fear of upsetting them. Yet, when a civilian becomes a soldier, a new factor arises, for not only is physical danger lurking near, but the specter of death may loom on the horizon. In certain people a central focus of fear is present. As a result of intensive and injudicious training such a person becomes aware of his heartbeat. The pounding of his heart at night, when alone in bed, prevents sleep; the condition may be aggravated by showers of extra systoles which are self-induced. A secret disturbance arises within the breast, at first only a tickle, gradually increasing in volume, and it only needs a shock to produce the whole psychological gamut and spate of symp-

toms known by various names—D.A.H., effort syndrome, cardiac neurosis, et cetera. Just like the bolting of a frightened horse or the running down of a piece of clockwork without its spring.

To recapitulate, the danger signals are: increased rate of pulse, increased awareness of the heart beat, reduplication at rest, sensation of throbbing in the head when lying or stooping, lassitude, and a tendency to become easily puffed. I would suggest that periodic medical inspections should be carried out in early training, so as to weed out the weak from the strong and the good from the bad, and to give the latter modified training. In that way I feel confident that breakdowns would be anticipated, and a large sum would be saved to the State in pensions. Finally, no one under the age of 18 years should be submitted to severe training. All athletic coaches are agreed on this.

SUMMARY

Reduplication after exercise is always of the first, and never of the second, sound, and reduplication of the first sound is an indication of the dilatation of the right ventricle. The right heart is the first to dilate as a result of exercise. Teleroentgenograms and accurate diagrams are given showing not only that the right side of the heart is the first to dilate as a result of exercise, but also the average increase. Teleroentgenograms have also been taken showing that the position of the heart in the thorax varies with posture, and the audibility of murmurs and reduplication in relationship to this is discussed. A detailed description of the pulse rates and blood pressures of boxers before and after contests is given. The effect of strain on the heart and its bearing on the training of the recruit is also described.

I must express my grateful thanks to the War Office and the L.C.C. for helpful cooperation; to Lord Horder for encouraging advice; to Drs. Parsons Smith and Daniel Davies for valuable help; to the x-ray staff of the County Hospital, Haverfordwest; and, in particular, to Mr. Weatherby, the radiographer.

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TRAUMATIC INJURY OF THE HEART

Incidence of Its Occurrence in Forty-Two Cases of Severe Accidental Bodily Injury

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ALTHOUGH numerous isolated cases of trauma of the heart are recorded in the literature, no investigation has, to my knowledge, been made as to the frequency of its occurrence in various accidental injuries of the body. Occasionally an author reports a series of several cases of cardiac damage due to trauma, but the causal relationship of the damage to the trauma is in many of these cases not definitely proved. Thus, Leinoff[1] reports twenty-two cases of cardiac injury resulting from accidents. Most of these, however, were first observed by the author many weeks, months, or even years, after an assumed accident, and the diagnosis of cardiac injury was based primarily on the history. Many of these were undoubtedly cases involving litigation, and the reliability of the patient's history may be questioned. Even if cardiac injury in such series of cases can be definitely proved to be due to trauma, they still do not give us any information as to the frequency of its occurrence in the usual run of accidental injuries of the body.

The first attempt to determine the incidence of cardiac trauma in bodily injuries was made by Barber.[2] He made electrocardiographic studies in thirty-three cases of chest injuries and found that eight of these showed abnormalities in the electrocardiogram. He selected only those cases in which the patient was less than 45 years of age, to exclude coronary disease as a possible cause for abnormal electrocardiographic changes.

Following my observations on trauma of the heart due to accidental injuries of the body in five cases previously reported,[3] I carried out a cardiac study in thirty-nine cases of accidental injuries of the body in which the patients were admitted to the surgical wards of the Coney Island Hospital, and in three cases from other sources. In addition to these forty-two cases, I observed nine other cases in which there was definite evidence of cardiac involvement which, according to the history, was traceable to accidental injury. Inasmuch as these nine patients were first observed many weeks or months after the accident, they are not included in the series.

The selection of cases was based on the degree of the accident and the extent of bodily injury. The cases were not limited merely to chest injuries, as was done by Barber. The reason is that, as has been repeatedly shown by various authors, and as I have described before,[4] trauma of the heart may occur as a result of a blow to any part of the body, provided it is of sufficient violence, and its force is transmissible to the heart. I have also included in the series individuals over 45 years of age, although coronary sclerosis might have been present in some. It is a known fact that, as I pointed out before,[4] injury to the heart caused by a blow may occur more readily and be more severe in individuals

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of the older age groups. I made sure, however, that in all cases selected there was no history of previous heart disease. In an occasional case where there was suspicion that cardiac disease might have been present before, I made sure that the patient did not show active symptoms and signs of such disease before the accident. Furthermore, only those patients who showed active progressive electrocardiographic changes soon after the accident were included. No cases were included in which there were static abnormalities in the electrocardiogram on repeated examination after the accident.

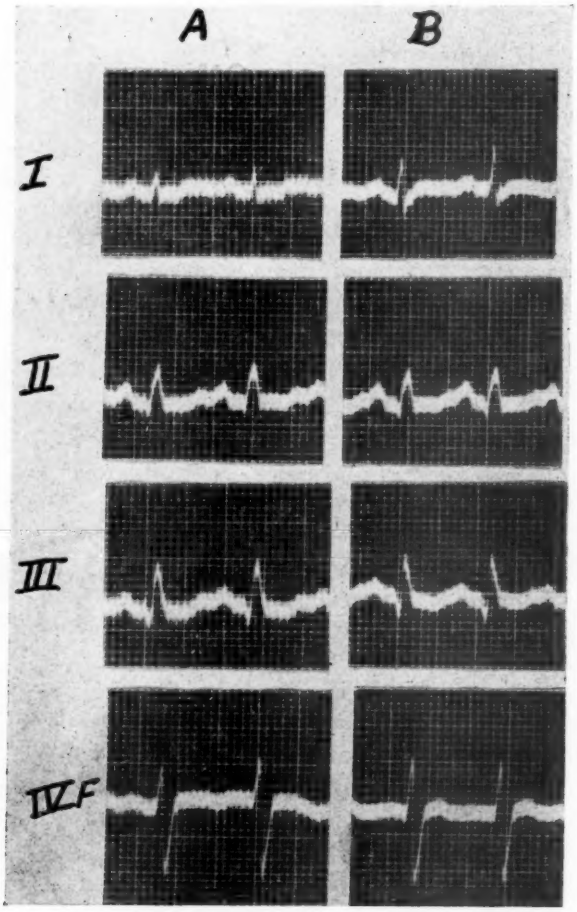


Fig. 1.—Electrocardiograms in Case 1. *A*, **taken day of accident;** *B*, **second day.**

The type of accident and the extent of general bodily injury were important guides in the selection of cases. Only those cases in which the injuries of the body were of sufficient degree to require hospitalization and in which the torsum was involved were chosen. Ambulatory patients with accidental injury were not studied.

Of the forty-two cases reported here, eleven are briefly reviewed and are accompanied by illustrations. The remaining thirty-one cases are summarized in Table I. The electrocardiographic changes of Cases 17, 18, and 19 in the table were presented in Figs. 182, 183, and 184 in the chapter on Trauma of the Heart previously alluded to.[4]

REPORT OF CASES

CASE 1.—A man, aged 41 years, with no previous history of heart disease, dived into the ocean and hit some object in the water. He fractured the right scapula, the left clavicle, the anterior part of the left second rib, and the frontal part of the skull extending to the base. He showed evidence of marked cerebral involvement. His heart was normal in size. The heart sounds were of very poor quality and a short gallop rhythm developed. He died two days after admission. The electrocardiograms in Fig. 1 were obtained on the day of the accident (*A*) and on the second day (*B*). Marked abnormalities are noted in the QRS complex and the T wave in both tracings, with progressive changes in configurations.

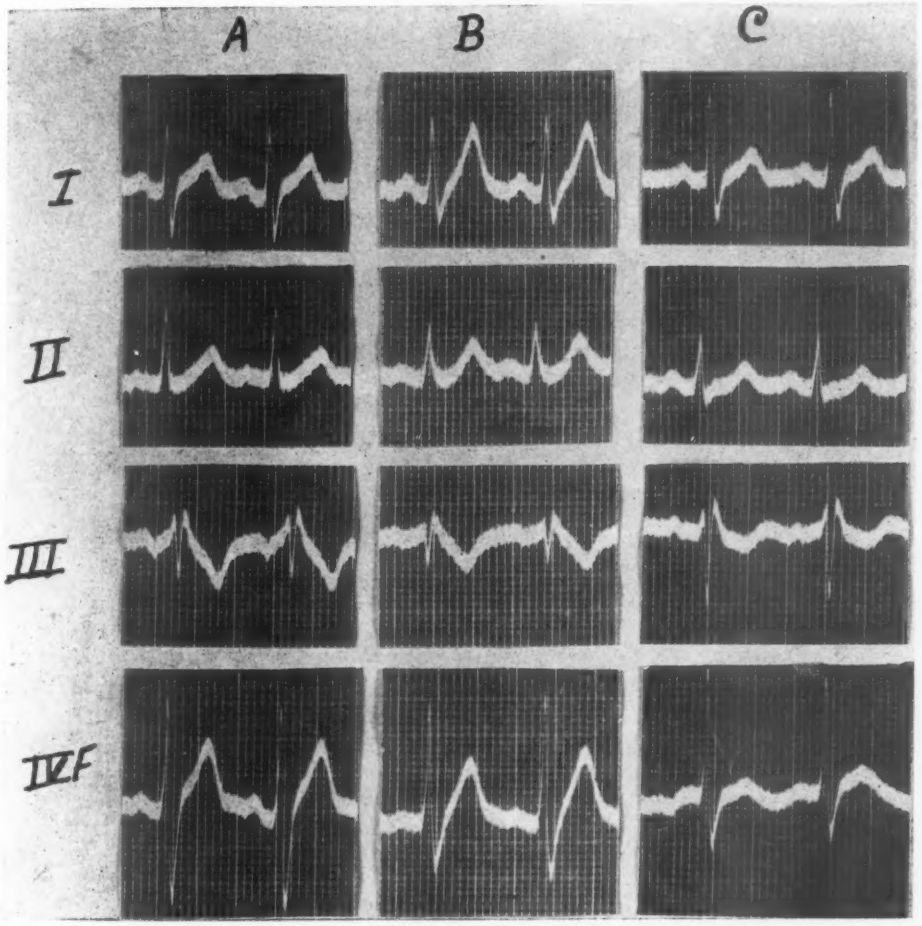


Fig. 2.—Electrocardiograms in Case 2. *A*, taken day of accident; *B*, one week later; and *C*, two weeks after *B*.

CASE 2.—An 18-year-old male with no history of any previous illness, jumped down the Marine Bridge at a height of about 30 feet. He sustained multiple contusions and abrasions of the body and fractures of the felt femur and the right radius. He ultimately developed osteomyelitis of the femur and died after nine months of confinement in the hospital.

Besides the general bodily pains, due to multiple injuries, he complained of precordial discomfort. His heart sounds were muffled and split at various times. A short pericardial friction rub was heard on the tenth day of admission. A short systolic murmur developed at the apex on the seventeenth day, which later disappeared. A short presystolic gallop rhythm was present at times. At no time did he show any signs of congestive failure.

Many electrocardiograms were obtained in his case before osteomyelitis developed. Fig. 2 represents three selected tracings illustrating some of the changes in the configurations of the complexes. Record *A* was obtained on the day of the accident. There is a large S

wave in Lead I, and a tendency to left axis deviation and an abrupt QRS-T takeoff with a markedly negative T wave in Lead III. Record *B* was obtained one week later. The S wave in Lead I is of lower voltage and broadened, while the T wave is markedly exaggerated. In Lead II the R wave is of lower voltage and the R-T segment is rounded. In Lead III the QRS complex is of lower voltage and the T wave is less negative. In Lead IVF, the S wave is of lower voltage and broadened, and the S-T segment is very abrupt. Record *C* was obtained two weeks after *B*. At this time there is a greater degree of left axis deviation with diminished voltage of the T wave in Leads I, II, and IVF. The T wave in Lead III is diphasic.

CASE 3.—A man, aged 40 years, was struck by an automobile and sustained fractures of the left radius and ulna, the right radius, the right tibia and fibula, the left fourth rib in the midaxillary line, and the acromium process of the left scapula. He had general bodily contusions and abrasions. In addition to generalized pains, he complained of precordial pain the first few days. His heart was not enlarged, but the first sound was diminished in intensity from time to time during the first two weeks. He recovered completely from all his injuries after three months confinement in the hospital.

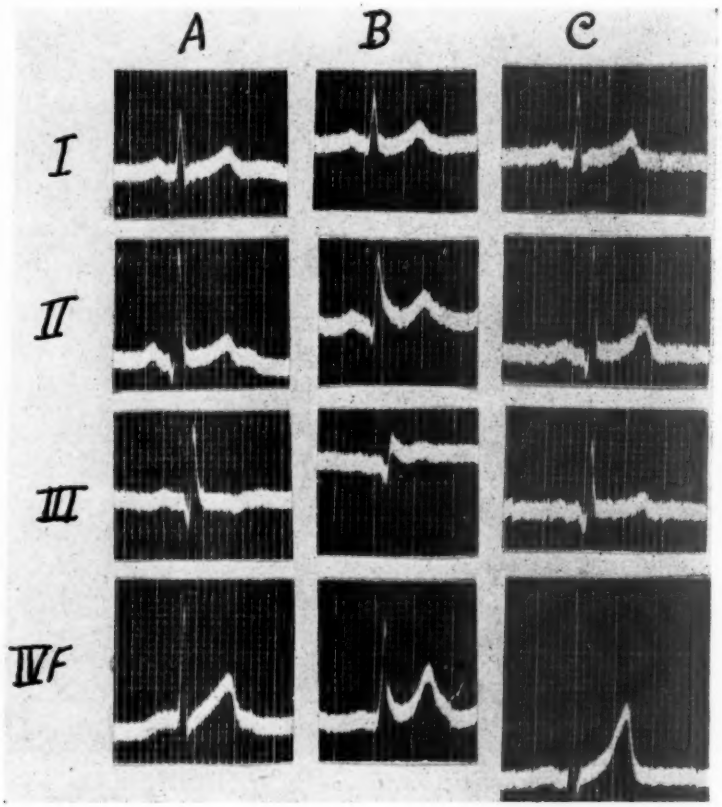


Fig. 3.—Electrocardiograms in Case 3. *A*, *B*, and *C*, taken two, fifteen, and eighteen days, respectively, after the accident.

Many electrocardiograms were obtained in his case, three of which are shown in Fig. 3. Record *A* was obtained two days after the accident. It is within normal limits. There is a large U wave present, shown particularly in Lead II. Record *B* was obtained fifteen days after the accident. The voltage of the R wave in all leads, especially in the third, is much lower, the R-T segment is rounded in all leads and elevated in Leads II, III, and IVF, and the T wave is of higher voltage in all leads, being positive instead of negative in Lead III. Record *C* was obtained eighteen days after the accident. The tracing is similar to that of Record *A* except that the T wave is of higher voltage in Leads I and II, positive in Lead III, and both the QRS complex and T wave are of much higher voltage in Lead IVF. The U wave is absent in all leads. Subsequent tracings obtained on him showed no further changes.

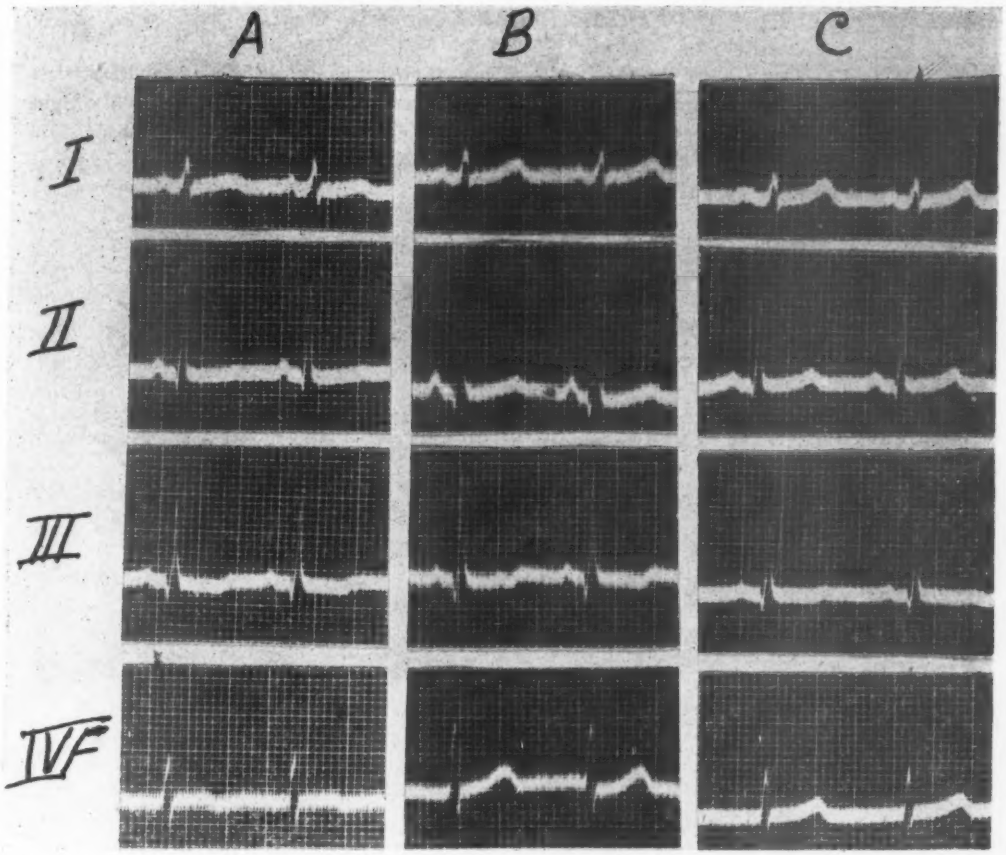


Fig. 4.—Electrocardiograms in Case 4. *A*, *B*, and *C*, taken several hours, two days, and twenty days, respectively, after the accident.

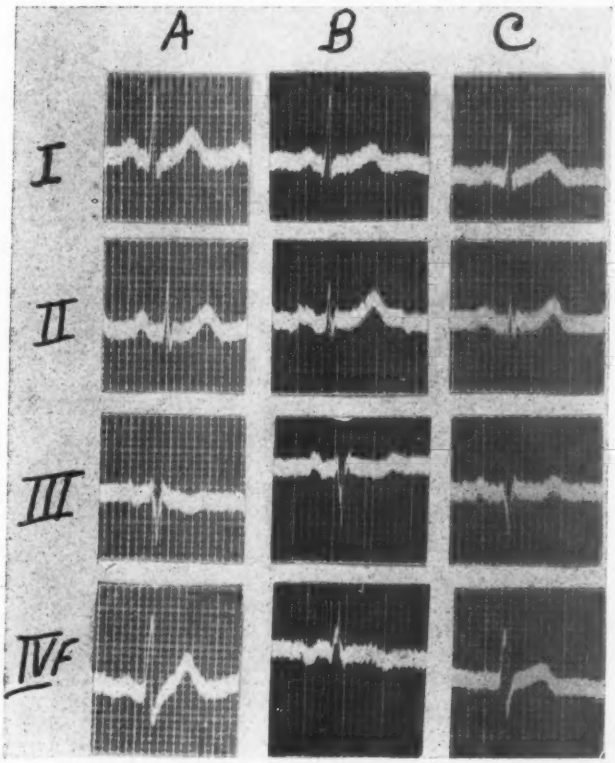
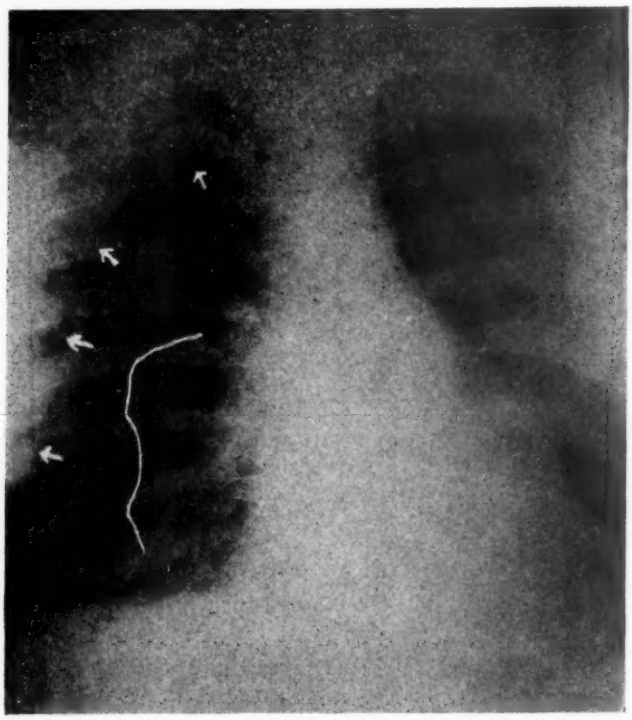
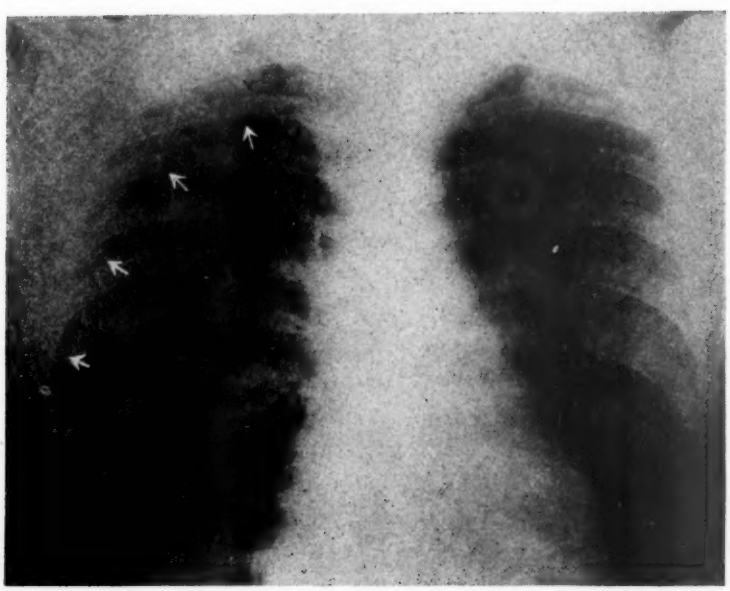


Fig. 5.—Electrocardiograms in Case 5. *A*, *B*, and *C*, taken one, four, and six days, respectively, after the accident.

CASE 4.—A man, aged 44 years, who was always well, was struck by a car and sustained fractures of the right tibia and fibula, multiple fractures of the pelvis, fractures of the right fifth and sixth ribs in the midaxillary line and contusions and abrasions of the body. He completely recovered after thirty-two days' stay in the hospital. There were no symptoms or abnormal physical signs of heart involvement.



A.



B.

Fig. 6.—Teleroentgenograms in Case 6. *A* and *B*, taken one and nine days, respectively, after the accident. Arrows point to the edge of the compressed lung by the pneumothorax.

The electrocardiogram (Fig. 4, *A*) was obtained several hours after the accident. The T wave is of low voltage in Lead I, almost isoelectric in Lead II, negative in Lead III, and of very low voltage, diphasic, in Lead IVF. Fig. 4, *B*, was obtained two days after the accident. The QRS complex is of higher voltage in Leads II, III, and IVF. The T wave is of higher voltage in Leads I, II, and IVF. Fig. 4, *C*, was obtained twenty days after the accident. Its appearance is the same as in *B* except that the QRS complex in Leads II, III, and IVF is now of the same voltage as in the corresponding leads in *A*. The relatively low voltage R wave in Lead I in this case is due to a longitudinally shaped heart which he presented.

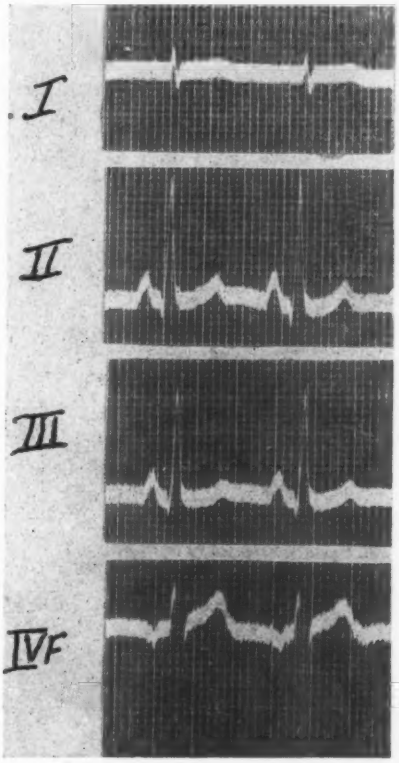


Fig. 7.—Electrocardiogram in Case 6.

CASE 5.—A woman, aged 49 years, was struck by a car and sustained contusions and abrasions of the body and lacerations of the face. There were no fractures of bones. She complained of general bodily pains and marked precordial oppression. There was tenderness over the left upper anterior chest. The size of the heart was normal, and the rhythm was regular, but the first sound was very short and of valvular quality. A faint systolic murmur was heard over the apex during the first two days. The heart sounds returned to normal after several days.

Fig. 5, *A*, depicts her electrocardiogram one day after the accident. It shows merely left axis deviation. Fig. 5, *B*, was obtained four days after the accident. The T wave in Lead I is of lower voltage. In Lead IVF, the P wave is negative, the R wave is of low voltage and markedly notched, the S wave is almost absent, the S-T segment is coved, and the T is negative. Fig. 5, *C*, was obtained six days after the accident. The R wave in Lead I is of lower voltage, the QRS complex in Lead II is multiphasic and of much lower voltage, and the appearance of the ventricular complexes differs from these of *A* and *B*.

CASE 6.—A man, aged 36 years was struck by an automobile and was brought to the hospital in a stuporous condition. He showed marked contusions and abrasions of the body. An x-ray film of his chest one day later (Fig. 6, *A*) shows right pneumothorax with collapse of about one-third of the right lung. The heart and mediastinum are deviated to the left. There are no fractures of any of the bones of the chest. In an x-ray film taken eight days

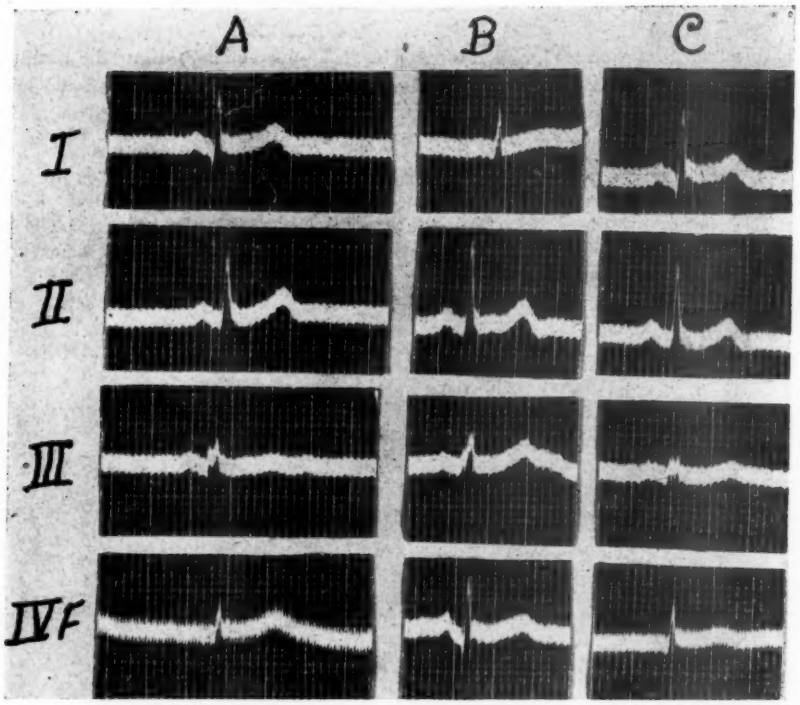


Fig. 8.—Electrocardiograms in Case 7. *A*, *B*, and *C*, taken one, four, and twenty days, respectively, after the accident.

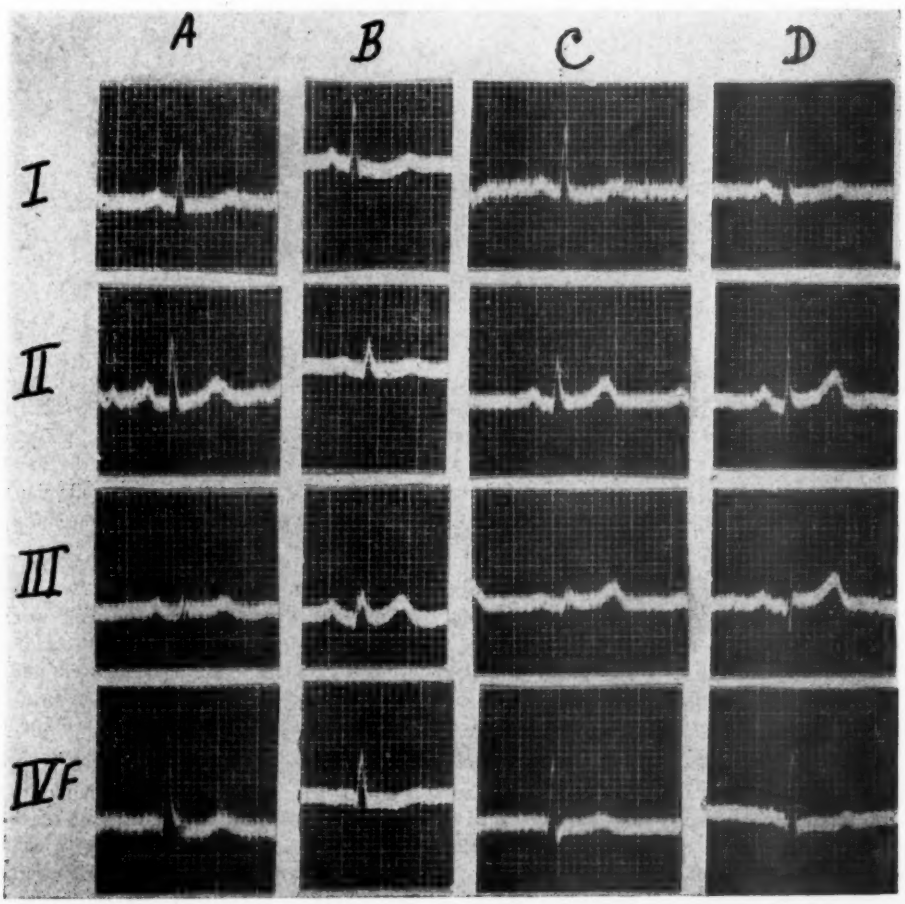


Fig. 9.—Electrocardiograms in Case 8. *A*, taken soon after the accident; *B*, *C*, and *D*, two, four, and six days later, respectively.

later (Fig. 6, *B*), the pneumothorax is slightly less marked. The heart sounds were of poor quality, and a short systolic murmur was heard over the aortic area.

Electrocardiograms, obtained on the second, third, fourth, fifth, and ninth day after the accident were alike, without progressive alterations; one is shown in Fig. 7. The low voltage ventricular complexes in Lead I are probably due to shift of the heart to the left, although the possibility that it may be due to structural damage of the heart is to be considered.

CASE 7.—A girl, aged 19 years, was thrown off the running board of a fast moving automobile. She sustained multiple contusions and lacerations of the body and a fracture of the skull. She made a complete recovery after a stay in the hospital of about ten weeks. Her heart showed no detectable abnormalities the first eight days. On the ninth day, she developed a loud systolic murmur between the third and fourth intercostal spaces, close to the sternum, and the heart rate increased from 72 per minute, in the recumbent posture, to 125, when she attempted to sit up. She had no subjective complaints referable to the heart. The murmur disappeared entirely after ten days.

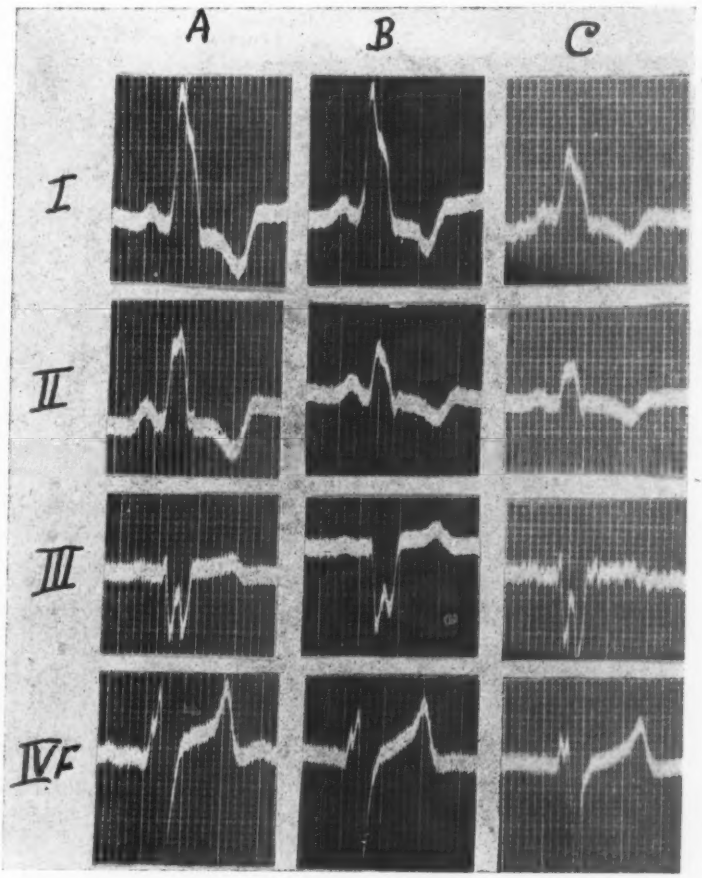


Fig. 10.—Electrocardiograms in Case 9. *A*, *B*, and *C*, taken two, four, and eleven days, respectively, after the accident.

In Fig. 8, *A*, *B*, and *C*, are three electrocardiograms obtained one, four, and twenty days, respectively, after the accident. In *A* the QRS complex in Lead IVF is of low voltage. In *B* the QRS and T waves in Lead I are of lower voltage than in *A*, while those of the other leads are of higher voltage than before. In *C* the findings are practically the same as in *A*.

CASE 8.—A woman, aged 62 years, was hit by an automobile and was thrown to the ground. She sustained a fracture of the left femur and marked contusions of the body especially the left side of the chest. No ribs were fractured. Her heart was slightly enlarged, the first sound was somewhat diminished in intensity, and the second sound at the aortic area

was accentuated. There was a rough systolic murmur over the aortic area, transmitted over a fairly large area, which was undoubtedly present before the accident and indicated pathologic changes of the aorta. Her blood pressure measured 200/100. She had moderate peripheral arteriosclerosis and the x-ray examination showed calcification of the abdominal portion of the aorta. She undoubtedly had considerable coronary sclerosis with myocardial damage before the accident.

Fig. 9, *A*, is a tracing obtained from her soon after the accident. The T wave in Leads I and IVF is of low voltage, and there is some depression of the R-T segment in the latter lead. Fig. 9, *B*, was obtained two days later. The voltage of the QRS complex in Lead I is higher, while in the other leads it is lower, than in *A*. There is some depression of the R-T segment in Lead I. The T wave is of lower voltage in Lead II, higher in Lead III, and lower in Lead IVF. Fig. 9, *C*, obtained four days after the accident, shows further changes, especially in Lead IVF, which now approaches a more or less normal appearance. The general appearance of the three leads is approximately the same as the corresponding leads in 9, *A*. Fig. 9, *D*, obtained six days after the accident, is about the same as 9, *C*, except for a somewhat higher voltage T wave in Leads II and III and the presence of a fairly large Q wave in Lead III.

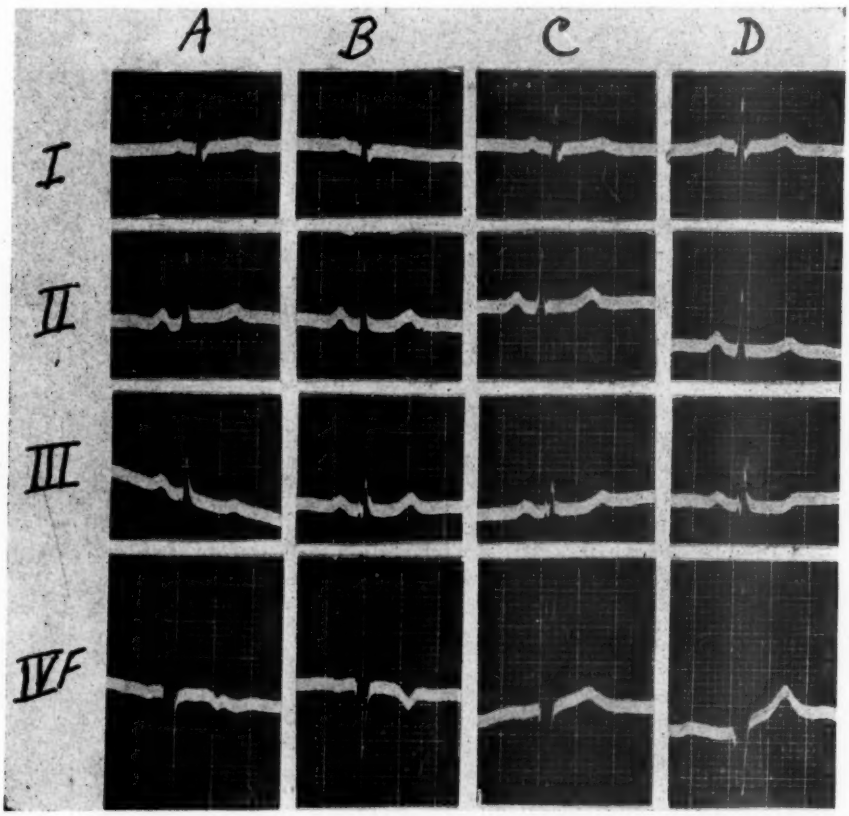


Fig. 11.—Electrocardiograms in Case 10. *A*, *B*, *C*, and *D*, taken twelve, twenty, twenty-eight, and thirty-six days, respectively, after the accident.

CASE 9.—A man, aged 67 years, fell on the ground and sustained a fracture of the left humerus, the left third, fourth, and fifth ribs, and contusions of the chest. He complained of pain in the injured areas, but there were no symptoms referable to the heart. He recovered completely. His heart was enlarged, and the sounds were of the type often heard in cases of bundle branch block. There was moderate congestion at the bases of the lungs. He had marked generalized arteriosclerosis.

An electrocardiogram (Fig. 10, *A*) was obtained two days after the accident. It shows left bundle branch block. Fig. 10, *B*, four days after the accident, still shows left bundle branch block, but some changes are noted in the configuration of the complexes, especially in

Lead II. Fig. 10, *C*, eleven days after the accident, shows further changes in the configuration of the ventricular complexes; left bundle branch block is still present. The bundle branch block probably existed before the accident, but the changes in the appearance of the complexes from time to time assume significance in indicating acute alterations in intraventricular conduction.

CASE 10.—A male, laborer, aged 51 years was excavating a ditch and was buried up to his neck by a heavy bank of ground which caved in. He sustained fractures of the pelvis and the free costal cartilages of the left ninth, tenth, and eleventh ribs. A day later, he began to experience retrosternal pain, radiating to the left shoulder, with choking sensation and a feeling of suffocation. This continued with greater or less severity for many weeks. His heart was not enlarged, and the sounds were accentuated. No murmurs were audible. An electrocardiogram, twelve days after the accident (Fig. 11, *A*), shows a low voltage T wave in all standard leads and a peculiar triphasic T wave in Lead IVF. Twenty days after the accident (Fig. 11, *B*), the T wave is isoelectric in Lead I, more positive in Leads II and III, and markedly negative in Lead IVF. Twenty-eight days after the accident (Fig. 11, *C*), the T wave in Leads I and IVF is positive but of low voltage, while in Leads II and III it remains the same. Thirty-six days after the accident (Fig. 11, *D*), the T wave is of higher voltage in Leads I and IVF.

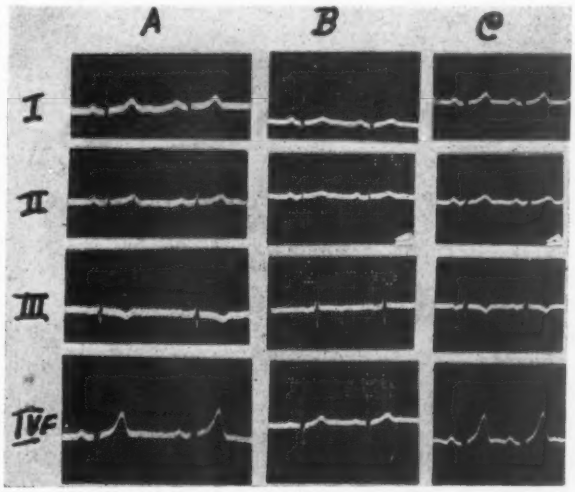


Fig. 12.—Electrocardiograms in Case 11. *A*, taken one and one-half years before the accident; *B* and *C*, six days and seven weeks, respectively, after the accident.

CASE 11.—A male physician, aged 46 years, while driving his car, collided head-on with another car and was thrown against the steering wheel. He sustained fractures of the second to the sixth right ribs at the costochondral junction with displacement. The angle of Louis became exaggerated, but there was no demonstrable fractures of the sternum. There was little shock but some pain was present in the sternal region on breathing or on moving around. He was admitted to a hospital where he stayed ten days. At the end of this time he felt well enough to return to his practice. While in the hospital, the heart sounds, as recorded by his attending physician, were normal, and repeated examinations failed to reveal any murmurs. An electrocardiogram he had obtained about one and one-half years before the accident (Fig. 12, *A*) shows a tendency toward left axis deviation. One obtained six days after the accident (Fig. 12, *B*) shows a definitely lower voltage T wave in all leads. Another tracing obtained about seven weeks after the accident (Fig. 12, *C*) is the same as the one before the accident.

About three weeks after the accident a loud systolic murmur was heard throughout the precordium by several physicians who examined him. I saw him for the first time about seven months after the accident when he presented a harsh systolic murmur occupying almost the entire systolic period with its maximum intensity at the third left intercostal space, close to the sternum, transmitted throughout the precordium, as far as the left anterior axillary line in the fifth space, and upwards as far as the right clavicle. The heart rate was 96 per minute and the rhythm was regular. The heart sounds were of good quality.

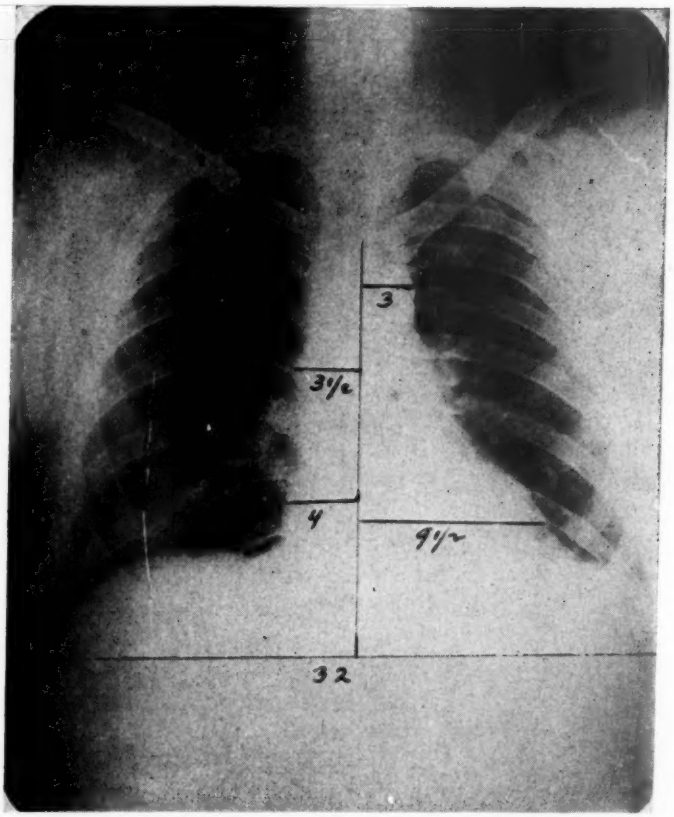


Fig. 13A.—Teleroentgenogram in Case 11, taken thirteen days after the accident. The figures represent the diameter in centimeters.

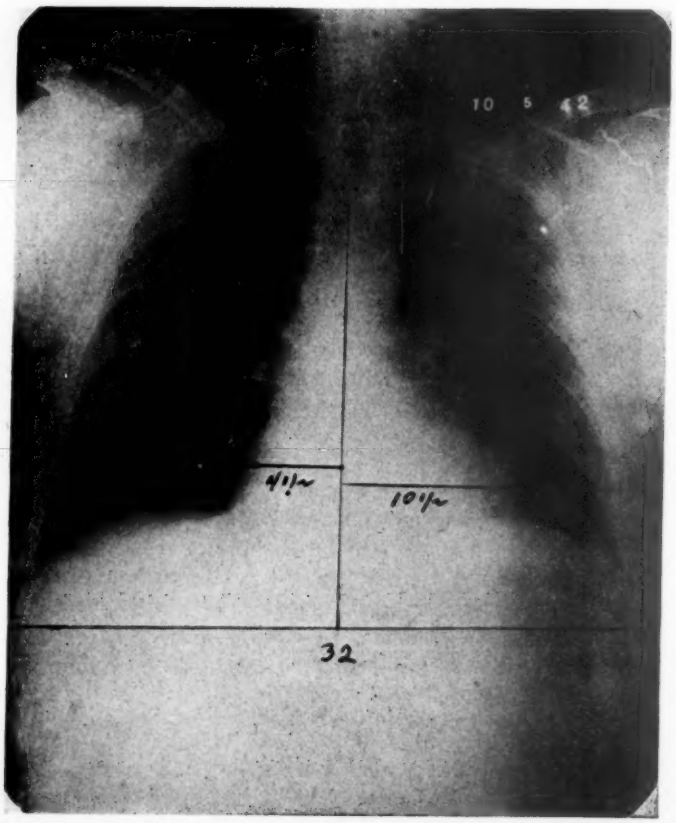


Fig. 13B.—Teleroentgenogram in Case 11, taken seven months after the accident. The figures represent the diameter in centimeters.

Inasmuch as the patient lives a great distance away from New York, I had no opportunity to observe him further. Frequent communications received from him, now nearly three years after the accident, indicate that he is able to continue his practice. He lives a comparatively strenuous life without any undue discomfort. His heart is moderately enlarged to compensate for an interventricular septal defect which he evidently developed as a result of the accident. The enlargement is shown in Figs. 13*A*, 13*B*, and 13*C*, obtained about thirteen days, seven months, and one year, respectively, after the accident.

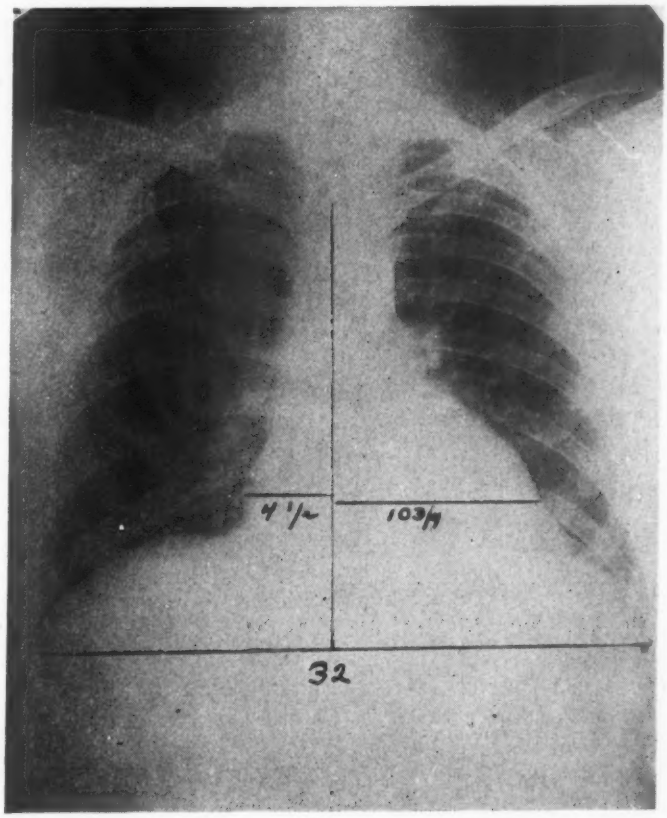


Fig. 13*C*.—Teleroentgenogram in Case 11, taken one year after the accident. The figures represent the diameter in centimeters.

DISCUSSION

It will be noted from the review of the forty-two cases reported here that cardiac damage occurs rather frequently in association with the general run of accidental bodily injuries, if the blow is of sufficient severity. In the forty-two patients, thirty-two, or 76.2 per cent, had demonstrable, cardiac injury, either clinically or electrocardiographically or both. Of these thirty-two patients, twenty-two had injuries of the chest wall, in addition to other bodily injuries, and, in ten others, the chest wall was not injured. Of those with chest-wall injuries, fifteen had fractures of one or more ribs and seven others had merely contusions and abrasions. If the degree of the blow to the chest can be measured by the presence or absence of fractures of ribs, we may assume that the blow to the chest in the seven patients without fractures was not marked, and the main force that injured the heart was transmitted from other parts of the body. In other words, in seventeen cases out of thirty-two with demonstrable cardiac injury, the transmitted force producing such injury came either entirely or mainly from other parts of the body.

TABLE I

CASE SEX AGE (YR.)	NATURE OF ACCIDENT	BODILY INJURIES	ABNORMALITIES OF HEART		
			DAYS AFTER ACCIDENT	SYMPTOMS AND ABNORMAL PHYSICAL FINDINGS	ELECTROCARDIOGRAMS
12 Male 53	Struck by automobile	Fracture of 8th, 9th, 10th, 11th, and 12th left ribs in anterior axillary line	1	Dyspnea, precordial pain. Normal findings	Normal
			2	Same	Slightly higher voltage R_2
			4, 7, 9, and 12	Same	Same as on second day
13 Male 28	Struck by bus	Basal fracture of skull, cerebral hemorrhage	2	Nothing abnormal	Left axis deviation
			3	Nothing abnormal	Less left axis deviation, large QRS, and T_4
			4, 5, and 7	Nothing abnormal	Same as 2 days after accident
14 Male 16	Fell off truck which was running at high speed	Fractured skull, brain injury	Same	None	Normal
			4	None	Bradycardia, T_4 much higher voltage
			7	None	Normal; same as on first day
15 Male 59	Struck by automobile	Comminuted fracture of tibia and fibula, followed by osteomyelitis, septic temperature, amputation and death 2½ months later	Same	None	Tendency, left axis deviation
			2	Poor first sound, systolic murmur at apex and pericardial friction rub at apex	T wave lower voltage in all leads
			6	Heart sounds better; no rub	Same as 2 days after accident
16 Female 57	Struck by automobile	Fractured pelvis, concussion of brain	19, 20, 26, 31, and 38	Enlarged heart, markedly diminished first sound, frequent premature contractions, systolic at base transmitted to apex	Low voltage T_1, tendency to left axis deviation, ventricular premature contraction, later T_1 isoelectric and T_2 and T_3 low voltage, T_4 negative
17 Female 18	Thrown off car at high velocity in amusement place	Severe body injuries, fracture right 11th and 12th ribs, rupture right kidney, liver injury, fracture right radius	1	Short gallop apex, systolic murmur over pulmonic area	T_1 isoelectric, T_2 and T_3 slightly diphasic, T_4 negative
			5	Same	T_1 slightly positive
			7 and 13	None	Normal
18 Male 50	Fell from second-story window	Fractured skull and multiple fractures left 3rd, 4th, and 5th ribs and left clavicle, shock	Same	Muffled, soft first sound, shock	T_1 low voltage, T_4 diphasic
			2	Precordial pain, muffled sounds	Very low voltage T, all leads
			12	Sounds better quality	R-T_1 and R-T_2 elevated, T waves normal in all leads
19 Female 40	Struck by automobile	Multiple fractures of pelvis, body contusions	1	None	Normal
			4	Epigastric and retrosternal pain, 2 days	T_3 and T_4 negative, changes in voltage of QRS
			6	Same	
			18	None	T_1 and T_2 lower, T_3 isoelectric, T_4
					Slightly positive. Normal
20 Female 70	Thrown to ground by bicycle	Fractured right radius and ulna	1	None	Normal
			8	None	Higher voltage in Leads I, II, and IVF
			14	None	Still higher voltage in Leads I, II, and III, lower IVF

TABLE I—CONT'D

CASE SEX AGE (YR.)	NATURE OF ACCIDENT	BODILY INJURIES	ABNORMALITIES OF HEART		
			DAYS AFTER ACCIDENT	SYMPTOMS AND ABNORMAL PHYSICAL FINDINGS	ELECTROCARDIOGRAMS
21 Male 58	Struck by automobile	Fracture of left tibia and fibula and 5th to 7th left ribs	1 10 20	None None None	Normal Lower voltage QRS with rounding of R-T segment, all leads Normal, same as one day after accident
22 Male 8½	Fell from a height of 12 feet, striking wooden fence	Laceration and large hematoma of liver at surgical operation	8 10	None None	None T_4 lower voltage
23 Male 28	Struck by automobile	Contusions and abrasions whole body, no fractures	2	Poor quality first sound	Negative T waves in Leads II, III, and IVF
24 Female 37	Fell down a flight of stairs	Fracture left femur, contusion of left chest and body	4 weeks	Precordial pain, dyspnea, weak sounds	Low voltage T wave, all leads, with slight depression of R-T segment
25 Male 13	Fell down while skating	Fracture left arm and contusions of chest	1 2	None None	Normal No change
26 Female 54	Hit by a man and fell to ground	Transverse complete intra-articular fracture right radius, multiple fracture of pelvis	2 3, 7, 11, 14, and 21	None, blood pressure 160/80 None	Normal No change
27 Male 3½	Fell 18 feet from window	Cerebrospinal injuries	8 and 10	None	Normal
28 Female 22	Struck by car, thrown to ground	Contusions of body, laceration of scalp	4 6, 13, and 17	None None	Rounding R-T_2 and R-T_3 Normal, no change
29 Female 14	Struck by automobile	Concussion of brain	2 and 3	None	Normal
30 Male 65	Struck by football player, fell	Fracture of greater trochanter	3 5, 6, 9, and 16	None None	Left axis deviation Lesser left axis deviation but no changes
31 Male 48	Fell down one flight of stairs	Contusions and abrasions of chest, sprain right ankle, cerebral concussion	2 3, 4, 6, 7, and 10	Precordial pain, diminished first sound Diminished pain, sounds normal	Normal Normal, slight change in T_3 from time to time
32 Female 47	Fell to ground	Fracture of right fibula	2 and 5	None	Tendency to left axis deviation, no changes
33 Male 57	Struck by automobile, fell on buttocks	Fracture shaft femur and fibula. Died 10 days later from pulmonary embolization	2, 4, 6, and 9	None	Left axis deviation
34 Male 41	Struck by automobile	Contusions and abrasions of body, fracture 9th and 12th right ribs, shock	Same 4 and 7	Chest pain, cough cyanosis. Greatly diminished first sound Sounds better. Generally better	Left axis deviation Left axis deviation, no change
35 Male 76	Struck by automobile	Contusions of left chest	Same 1, 3, and 9	Chest pain. Split first sound Chest pain. Split first sound	Normal No change

TABLE I—CONT'D

CASE SEX AGE (YR.)	NATURE OF ACCIDENT	BODILY INJURIES	ABNORMALITIES OF HEART		
			DAYS AFTER ACCIDENT	SYMPTOMS AND ABNORMAL PHYSICAL FINDINGS	ELECTROCARDIOGRAMS
36 Male 12	Struck by automobile	Fracture left tibia and fibula and right tibia	2	None	Normal
			8	None	Normal but lower voltage complexes
			11	None	Normal
37 Male 30	Struck by automobile	Injury left chest; fracture neck of 3rd left rib	1 and 6	Precordial and left chest pain. Diminished first sound	Normal
38 Male 40	Auto collision, thrown against steering wheel	Fracture right 3rd rib, secondary pleurisy	Same	Sounds muffled	Tendency left axis deviation
			2, 4, and 6	Sounds gradually improved	Lower voltage QRS
39 Female 44	Struck by automobile	Compound fracture left tibia and right fibula, contusions of body	2	Precordial pain. Accentuated first sound, short gallop	Tendency left axis deviation
			6, 9, and 21	Return to normal sounds	Gradual increase of left axis deviation
40 Male 57	Struck by automobile	Fracture of femur, severe contusions and lacerations of chest and face	2	None	Normal
			16	None	Same
41 Male 25	Fell on ground, striking chest	Contusions of chest, no fractures	1 to 3	Precordial pain fainting	None obtained
			4	Severe attack of retrosternal pain several hours	T_1 and T_4 negative
42 Male 52	Driving bus, collided with streetcar. Thrown against steering wheel	Contusion of chest	Same	Collapse, cold clammy perspiration, excrutiating precordial pain	None obtained
			3	Continuous pain. Faint heart sounds	Left axis deviation very low voltage QRS
			15	Signs of left ventricular failure	T_2 and T_3 negative. Left axis deviation, T_1 negative, T_2 slightly positive, T_4 negative

It is interesting to find, as illustrated in Case 6, that injury to the lung may also occur without any damage to the chest wall. That is, under certain circumstances, a transmitted force from distant parts of the body may also produce other intrathoracic injury than that of the heart.

How trauma of the heart is produced by a blow to distant parts of the body cannot be easily explained. To my knowledge, no experimental studies of this problem have, as yet, been carried out. In the studies of the relation of heart injury to trauma carried out by Schlomka,[5] Kulbs and Straus,[6] Bright and Beck,[7] Kissane, Fidler, and Koons,[8] Moritz and Atkins,[9] and Randles, Gorham, and Dresbach,[10] injury of the heart was produced by direct blows to the chest or by blows to the exposed heart. No study has been made of the possible production of cardiac injury by severe blows applied to other parts of the body.

There are, however, many reports of isolated clinical cases of cardiac injury and rupture following an accidental blow to distant parts of the body. The cases of Howat,[11] Saphir,[12] Kampmann,[13] Kienle,[14] and Smith and McKeown[15]

are only a few of the many found in the literature. It is undoubtedly the suddeness of the blow applied to the body and transmitted to the heart by the severe vibrations set up which results in the injury. It is possible that, in some cases, the damage of the heart is not produced directly by the transmitted force on the heart muscle but by coronary spasm induced by the blow, resulting in myocardial ischemia. This theory was postulated by Schlomka[5] in his experimental work and may also apply to the human being. The subject has been described in my previous communication.[4] The factor of shock, with its emptying effect on the coronary system, also may play a part.

The cardiac injury in our series was usually manifested soon after the accident. In some cases there was a delay of one or more days before symptoms and signs or electrocardiographic evidence developed. In many of these cases the clinical and electrocardiographic manifestations were very mild in degree and short-lived so that they could easily have been missed if frequent examinations had not been made.

The subjective manifestations were precordial discomfort in one case; precordial pain in nine cases; precordial pain together with dyspnea in two; retrosternal and epigastric pain in one; retrosternal pain radiating to the left shoulder, together with choking sensation in one; and chest pain, cyanosis, and cough in another. Seventeen patients had no subjective complaints. In general, the subjective complaints suggestive of cardiac injury were mild compared with complaints referred to other bodily injuries.

The objective manifestations consisted of changes in the character of the heart sounds and the presence of a gallop rhythm, a pericardial friction rub, murmurs, and premature contractions.

The changes in the character of the heart sounds were usually short-lived. In eleven cases, the first or first and second sounds were diminished in intensity. In three cases the sounds were muffled. In one the first sound was split, in another it was of valvular quality, and in two the sounds were accentuated. In three cases a gallop rhythm developed, lasting a few days, and in a fourth case a gallop rhythm was associated with marked diminution in the intensity of the first sound. A pericardial friction rub occurred in two cases and was fleeting.

The murmurs heard were all systolic in time. In three cases it was heard at the apex with a comparatively small area of transmission. In three cases it was heard at the aortic area, but there was good reason to feel that in two (Cases 8 and 16) it was present before the accident and was due to pre-existing, atherosclerosis of the aorta. In one case the murmur was heard at the left sternal border, between the third and fourth intercostal spaces, and in one over the pulmonic area. In Case 11, the maximum intensity of the murmur was in the third left intercostal space with wide transmission, typical of the so-called Roger's murmur which goes with interventricular septal defect. Premature contractions occurred in one case.

The electrocardiographic changes were not specific and varied with different cases. The characteristic findings were frequent alterations in the configuration of the various deflections from day to day, or in the course of days in each case, as described before.[4] The changes consisted of occasional alterations in the P wave; changes in the direction of the electrical axis from time to time; and alterations in QRS complex, in the RS-T segment, and in the T wave.

The changes in the QRS complex consisted of variations in the heights of its various components from time to time, the development of slurring and notching

in occasional cases, and the appearance or disappearance of some of the waves of the complex.

The changes in the RS-T segment often consisted of rounding, elevation, and upward concavity in one or more leads seen in pericardial involvement. In occasional cases there was some coving as seen in myocardial infarction. In most cases the changes were nonspecific.

The T-wave abnormalities consisted of diminution in its height and change in its direction from positive to isoelectric and negative in various leads from time to time.

The pathologic changes that occur in the heart in trauma are probably the same as in the experimental animal, reported by the observers previously alluded to[5-10] and were fully described before.[4]

The outcome of traumatic injury to the heart in this group of cases was generally good. Complete recovery apparently occurred in nearly all cases. This was undoubtedly due to prolonged rest in bed which the other bodily injuries called for. In Case 1, in which the patient died, the cause of death was at least partly due to the cardiac injury, which was very marked. In Case 2, the patient died after many months from osteomyelitis, not primarily from his cardiac condition. In Case 11, the damage is permanent as judged by the persistance of signs, now, three years after the accident.

In a previous communication[3] I reported one case of traumatic injury of the heart which was observed for more than two years. At the end of this time the patient showed complete clinical recovery, but Lead IVF in the electrocardiogram still showed abnormalities. In another case the patient showed evidence of severe myocardial and pericardial disease with calcification and auricular flutter, three and one-half years after an accident. One year later, he died in heart failure. A third patient still shows evidence of extensive organized posterior wall infarction with the anginal syndrome, now over four years after an accident.

Some years ago,[16] I reported an unusual case of left-sided displacement of the heart which I considered at that time to be probably of congenital origin. The patient, however, gave a history that, as a child, he fell from a window three stories high, and was bedridden about three months. In the light of subsequent experiences, I must agree with Bramwell and King,[17] who think that the abnormality in this case was probably caused by traumatic injury rather than due to a congenital defect. King reported two more somewhat similar cases, one his own and another by Howard, in both of which trauma, resulting in massive pleuropulmonary pericardial adhesions, was the cause of retraction of the heart to the left.

Although the number of cases presented in this paper is small, the frequency of occurrence of cardiac injury in this series is very significant. Such injury would certainly have been overlooked if no special investigation were made. The complaints of the patient referable to the heart were comparatively trivial and could have easily been missed by the surgically minded doctor.

The problem of trauma of the heart in bodily injuries certainly calls for further unbiased investigation, not colored by preconceived ideas. The study should include careful clinical, electrocardiographic, and other laboratory follow-up, of all severely injured patients, from the day of injury through recovery. Those that show some permanent defect of the heart should be observed for years to ascertain the ultimate outcome. The statement by Stroud,[18] "I

still feel cardiac injury is unusual following anterior chest trauma," frequently expressed also by other cardiologists, is certainly based on nothing more than belief.

SUMMARY

A study was made of the incidence of trauma of the heart in forty-two cases of rather serious accidental injuries to the body. In thirty-two, or 76.2 per cent, there was demonstrable evidence of some cardiac damage, clinical, electrocardiographic, or both. In some cases the damage occurred even if the blow did not affect the chest, provided it was of sufficient severity to result in marked bodily injury. In most cases the damage appeared to be very mild and short-lived. In some it was severe.

In many cases there were either no subjective manifestations referable to the heart or the manifestations were trivial. Precordial pain or discomfort and slight dyspnea were the main complaints. The objective manifestations consisted of abnormalities in the heart sounds, the presence of a gallop rhythm, a pericardial friction rub, and systolic murmurs, in various areas. The findings were short-lived and occurred in comparatively few cases.

The electrocardiographic manifestations consisted of changes in the voltage and appearance of the QRS complex from time to time and some shift in the electrical axis; elevation, rounding with upward concavity, and occasional coving of the R-T segment; and frequent changes in the T wave from positive to isoelectric and negative and back to positive. In the majority of cases the electrocardiographic changes were nonspecific. In occasional cases, they were characteristic of pericardial involvement, or of localization of the damage to other specific areas of the heart.

Complete recovery took place in nearly all cases in this series. Only one patient showed permanent damage and in another case the cardiac injury was at least partly responsible for death.

The observations tend to indicate that trauma of the heart is a rather frequent occurrence in serious bodily injuries and call for future unbiased investigation of this subject.

I am indebted to Drs. D. A. McAteer, J. E. Hammett, J. E. Miles, and G. Webb, for the privilege accorded me of carrying out the observations presented in this paper on patients from their surgical services.

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STUDIES CONCERNING THE ETIOLOGY AND PATHOGENESIS OF NEUROCIRCULATORY ASTHENIA

III. THE CARDIOVASCULAR MANIFESTATIONS OF NEUROCIRCULATORY ASTHENIA

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INTRODUCTION

IN PRECEDING articles,[1, 2] the nature and pathogenesis of hyperthermia, giddiness, and syncope, found in the syndrome of neurocirculatory asthenia (NCA), were discussed. As a result of these studies, attention was directed toward the hypothalamus as a possible factor in the pathogenesis of the somatic phase of this illness. In the present communication, the results of clinical, physiologic and pharmacologic studies of the cardiovascular manifestations of neurocirculatory asthenia are reported.

In the past, there has been considerable disagreement concerning the exact status of the cardiovascular system in the patient with NCA. Some investigators[3-6] have not been able to detect any significant, or persistent, defect in the hemodynamics of these patients, but others[7-10] have reported abnormalities in either the size of the heart, in its function, or in its conduction system. It is worthy of emphasis, too, that whereas most internists[4, 8, 11] have consistently stressed the psychic factors in this disease, they have not succeeded in integrating the latter in any exact, physiologic manner with the actual emergence of cardiovascular symptoms and signs in the same patient. Likewise, the psychiatrists have not succeeded in elucidating the pathogenesis of cardiovascular manifestations in patients suffering from an obvious anxiety neurosis. There exists, then, a physiologic or neurologic void between the psychic and cardiovascular phases of neurocirculatory asthenia which has not been probed sufficiently by either the internist or by the psychiatrist. Until this void is explored, however, it will be impossible to understand those processes set loose in a person subject to anxiety, which express themselves in trembling, perspiration, flushing, dyspnea, palpitation, and precordial pain.

In an attempt to investigate the physiologic connection between the admitted emotional turmoil of the patient with NCA and his cardiovascular symptoms and signs, a study was made of fifty young soldier patients suffering from this syndrome. In this investigation, the hemodynamics of the patient with NCA

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were not studied solely as the functional expressions of isolated tissues containing and propelling blood, but rather as reactions of the entire cardiovascular system, a system intimately connected to the remainder of the body and controlled by nerve and by chemical reactions. For it is clear that any study of the heart and the organs associated with it *demands* such correlation. Otherwise, there is danger of grave inaccuracy, despite the use of the most precise devices designed to measure isolated hemodynamic functions.

A. PHYSICAL EXAMINATION OF THE HEART

Several workers[12, 13] have reported a small incidence of actual valvular heart disease in their series of patients with NCA. I was unable, however, to detect a single case of either congenital or infectious heart disease in my series. Inconstant, systolic, apical murmurs were heard in ten patients (20 per cent), but they lacked the identifying characteristics suggestive of damaged valves. Four patients (8 per cent) exhibited sharp, forceful heart beats against relatively thin chest walls.

B. THE SIZE OF THE HEART (ROENTGENOGRAPHIC DETERMINATION)

Although Master[7] has reported that the heart of the patient with NCA may be abnormally small, other investigators[3, 5] have found no particular abnormality in the size of this organ. Teleroentgenograms of the hearts of twenty patients with NCA and of fifteen normal young adults were obtained, measured, and compared. It was found that the average cardiothoracic ratio of the patients with NCA was 0.407 (range: 0.330 to 0.450) and that of the controls was 0.402 (range: 0.335 to 0.440). Thus, no abnormality in the size of the heart was found in the average patient with NCA in the present series. Furthermore, no abnormalities were found in the size or contour of the separate chambers of the heart.

C. ELECTROCARDIOGRAPHIC STUDIES

Both Master[7] and Merritt[10] have reported abnormalities of either the QRS complex or of the T wave in electrocardiograms obtained from patients with NCA. I was unable, however, to detect any significant, fixed abnormality in electrocardiograms of thirty patients with this syndrome. Nor was I able to confirm Master's observation that there is frequently a right axis deviation in the electrocardiogram of such patients.

In sharp contrast to the observations of other investigators,[4, 12, 14] I found that eleven of my patients (22 per cent) exhibited some form of transient arrhythmia during their hospital stay. Four had bouts of paroxysmal auricular tachycardia, five exhibited ventricular extrasystoles, one had transient episodes of auricular flutter, and one exhibited a wandering auricular pacemaker. The high incidence of arrhythmia in my series of patients might have been due to the fact that, besides obtaining routine electrocardiograms on admission, I also obtained additional ones when the patients informed me that they were experiencing palpitation. This last procedure was undertaken because it allowed the observation of the patient at the exact instant he was aware of heart dysfunction and enabled me to check in an objective fashion what he felt subjectively.

Two of the four patients with NCA who suffered from attacks of auricular paroxysmal tachycardia as frequently as five times a day were observed on several occasions before, and during, a bout of paroxysmal tachycardia. Invariably it was found that the onset of the arrhythmia was preceded by marked accentuation of the hand tremor, elevation of the oral temperature to hyperthermic

levels (99.2° to 100° F.), perspiration of the axillary and palmar skin, facial pallor, and a feeling of nervousness or tension. These symptoms and signs continued as the tachycardia began and persisted for a short while after the tachycardia had been interrupted by pressure over the carotid sinuses. It was found, too, that, when these same patients were given quinidine, their attacks of arrhythmia ceased, but they continued to experience and show, at intervals, the symptoms and signs previously described. This separation of the prodromal syndrome and the tachycardia itself, following the administration of quinidine, strongly suggested that the tachycardia observed was not the cause, but the result, of the peculiar nervous discharge which was seen to precede and accompany it.

D. OBSERVATIONS CONCERNING EXERCISE TESTS IN THE EVALUATION OF CARDIOVASCULAR EFFICIENCY

There has been some disagreement about the value of exercise tests in the evaluation of the patient with NCA. Several observers[4, 13] report that such a patient may show abnormalities in pulse and respiratory rates following such tests whereas other investigators[3, 6, 12, 15] have insisted that the usual exercise tests are of no value in detecting, or assessing, cardiac derangement in this type of patient. In connection with cardiac efficiency, the observations of Starr[9] are of interest in that he found some patients with NCA who exhibited hypofunction of the heart and others who showed hyperfunction.

TABLE I. THE RESPIRATORY RATE, PULSE RATE, AND BLOOD PRESSURE OF PATIENTS WITH NCA AND NORMAL ADULTS BEFORE AND AFTER A STANDARD EXERCISE TEST

CASE	BEFORE TEST			IMMEDIATELY AFTER TEST			TWO MINUTES AFTER TEST		
	RESP.	PULSE	B.P.	RESP.	PULSE	B.P.	RESP.	PULSE	B.P.
Patients With NCA									
1	20	80	135/72	28	140	170/80	28	104	120/80
2	20	96	130/95	36	150	155/90	20	108	135/95
3	20	100	115/75	48	130	150/70	34	100	135/75
4	18	118	110/80	28	156	140/75	20	138	115/80
5	20	96	145/100	36	140	210/90	24	126	170/90
6	20	72	120/75	18	84	130/75	20	74	120/65
7	20	84	130/75	34	120	135/70	26	96	130/70
8	18	112	120/80	36	144	130/70	22	118	120/80
9	24	96	135/78	32	144	160/60	32	114	155/70
10	18	88	120/70	48	168	135/70	24	114	120/80
11	20	64	135/80	36	120	170/70	42	68	145/80
12	36	88	110/70	42	168	135/65	36	98	110/70
13	24	104	110/75	36	144	130/70	30	96	110/70
14	20	80	115/80	36	160	135/80	24	120	115/80
15	20	90	120/85	24	156	145/75	24	114	135/80
16	18	68	115/70	30	120	130/70	18	60	120/70
17	20	76	135/80	24	132	180/70	21	90	135/60
18	18	78	135/80	24	120	150/70	18	80	140/75
19	20	84	130/90	22	132	140/90	20	84	130/90
20	28	104	120/80	36	192	165/70	32	120	130/80
21	24	88	115/80	38	168	155/70	30	108	125/70
22	18	100	115/75	30	192	125/65	20	102	120/70
Average	21	89	123/79	33	144	148/73	26	101	128/80
Normal Adults									
1A	16	76	110/75	32	120	130/65	18	72	110/70
2A	20	88	120/80	24	132	155/65	24	90	130/75
3A	14	84	95/55	20	144	120/50	14	80	100/60
4A	24	80	110/80	30	120	140/90	24	78	135/75
5A	18	84	120/80	18	144	165/55	18	76	135/75
6A	20	88	125/70	24	120	145/80	20	72	125/75
7A	18	84	125/85	24	132	150/70	24	94	125/65
8A	28	72	125/70	24	120	145/60	20	76	120/70
9A	20	78	125/65	25	180	165/55	21	81	124/65
Average	20	82	117/73	24	135	146/65	20	80	124/71

In our own efforts to determine the cardiac efficiency of the patient with NCA, we first employed a standard exercise test in which the patient jumped up and down (both feet together) one hundred times in sixty seconds. Measurements of the blood pressure, pulse, and respiratory rates were obtained before, immediately after, and again two minutes after, the performance of the test. Twenty-two patients with NCA and nine normal adults were studied. As Table I indicates, the resting values of the two groups were approximately the same. Other than excessive tachypnea in the patients with NCA, the values were also similar immediately after the performance of the test. When, however, the measurements taken two minutes after exercise were compared, it was found that the average pulse and respiratory rates of the patients with NCA were abnormally high.

On superficial analysis, the results above might seem to indicate a relative cardiac inefficiency in neurocirculatory asthenia. However, as mentioned before, it would be a mistake to assume, on the evidence of a persisting tachypnea and tachycardia alone, that these patients possessed an inadequate cardiovascular system. Other factors might have been responsible for the persistent tachycardia and the tachypnea. For example, it was found that quite frequently the patient with NCA, at the very beginning of the test, exhibited a tachypnea which was out of all proportion to the amount of work done up to that time. Accordingly, before the heart could be ascribed as the primarily deranged organ and the cause of the persisting tachypnea and tachycardia, the following observations had to be performed: (1) the effect of voluntary tachypnea on the pulse rate, (2) assessment of cardiac efficiency by other methods during the period of tachypnea and tachycardia after the standard exercise, and (3) the determination of the pulse and respiratory rate in an exercise test in which the possibility of anxiety on the part of the patient was eliminated.

1. *The Effect of Voluntary Tachypnea on the Pulse Rate.*—Seven patients with NCA and seven normal young adults were instructed to breathe deeply thirty times in forty seconds after their resting pulse had been taken. It was found that the patients with NCA showed an average increase of 30 beats per minute and the control group exhibited an average increase of five beats per minute after the hyperventilation had been completed. Four of the seven patients with NCA also exhibited a pounding heart and complained of palpitation. One also complained of precordial pain. These results indicated that tachypnea itself could induce an abnormal response in the cardiovascular functions of the patient with NCA. It suggested, too, that the high pulse rate observed in such a patient after the completion of the standard exercise test could have been caused by the tachypnea—a tachypnea which occurred so near to the onset of the test that it could not have been due to cardiac insufficiency.

2. *Assessment of Cardiac Efficiency During Standard Exercise Test by Measurements of the Vital Capacity, Venous Pressure, and Circulation Time.*—The vital capacity of seven patients with NCA was taken before and immediately after the performance of the standard exercise test. As Table II shows, the average vital capacity of the patient with NCA before and after exercise was 2.31 and 2.27 liters per square meter of body surface, respectively. The vital capacity, in other words, was within normal limits both before and after the test, and, after the latter time, the patient still exhibited a tachypnea and tachycardia. The venous pressure was measured in six of these patients by direct puncture of the right antebrachial vein, both before and immediately after exercise. It was found to average 6.6 cm. of water before, and 6 cm. of water directly after exercise. Both of these values are within normal limits. The circulation time (arm-

to-tongue) was determined by the injection of calcium gluconate into six patients with NCA, before, and two minutes after, the cessation of exercise. The average time of circulation before and after exercise was 12 and 10.5 seconds, respectively. Thus, it was found that despite the tachypnea and tachycardia of the average patient with NCA after performing the standard exercise test, there was no indication from these measurements that cardiac failure or inefficiency of any perceptible degree was present.

TABLE II. THE VITAL CAPACITY, VENOUS PRESSURE, AND CIRCULATION TIME OF PATIENTS WITH NCA BEFORE AND AFTER A STANDARD EXERCISE TEST

	BEFORE TEST			AFTER TEST		
CASE	VITAL CAPACITY*	VENOUS PRESSURE†	CIRCULATION TIME‡	VITAL CAPACITY	VENOUS PRESSURE	CIRCULATION TIME
1	2.25	5.5	12	2.25	4.0	10
2	2.70	10.0	12	2.70	11.0	10
3	2.50	5.7	12	2.55	6.2	11
4	2.00	2.0	13	2.14	3.0	12
5	2.40	9.5	12	2.45	6.0	10
8	1.80	7.0	13	1.70	6.0	10
9	2.53	—	—	2.11	—	—
Average	2.31	6.6	12	2.27	6.0	10.5

*Liters of air per square meter of body surface.
†Cubic centimeters of water.
‡Seconds.

3. *The Pulse and Respiratory Rate During Special Exercise Test.*—A simple test was devised which consisted of a small fixed pulley, over which a cord, fastened to an 8-pound weight, was suspended. The patient was seated and asked to raise and lower the weight over a distance of 10 cm., fifty times a minute, by pulling and releasing the cord. The pulse and respiratory rates were obtained at the end of 30, 60, 120, 180, 240, and 300 seconds. Before the patient was allowed to perform this test, however, he first watched others do it. He then performed the test three times on three different days; the last test was used for the calculations shown in Table III. The procedure was adopted in order to eliminate, as far as possible, any anxiety or nervousness on the part of the patient while performing the test. It was noted repeatedly that many patients with NCA began to breathe fast, with corresponding tachycardia, when they first attempted this simple exercise. Indeed it is felt strongly that much of the work

TABLE III. THE RESPIRATORY AND PULSE RATE OF PATIENTS WITH NCA AND NORMAL ADULTS BEFORE AND DURING A SPECIAL EXERCISE TEST

BEFORE TEST			DURING TEST									
			30 SECONDS		60 SECONDS		120 SECONDS		180 SECONDS		240 SECONDS	
CASE	RESP.	PULSE	RESP.	PULSE	RESP.	PULSE	RESP.	PULSE	RESP.	PULSE	RESP.	PULSE
Patients With NCA												
B1	20	80	20	96	22	90	22	90	22	102	24	108
B2	22	88	20	102	24	108	26	102	24	112	24	115
B3	18	78	20	98	20	102	22	98	24	102	24	102
B4	19	78	20	90	20	102	20	98	20	102	22	102
B5	21	60	22	66	24	72	22	84	22	84	22	—
B6	22	88	22	102	22	104	24	106	22	120	23	120
B7	18	78	18	84	20	90	22	96	24	96	22	—
B8	19	88	18	100	20	96	20	102	22	102	22	120
Average	20	80	20	92	22	96	22	97	23	103	24	111
Normal Adults												
C1	18	78	20	96	22	96	24	90	24	92	24	98
C2	21	78	20	94	24	104	24	104	22	108	22	114
C3	19	80	22	90	22	98	24	102	26	108	24	114
C4	16	72	18	76	20	84	20	84	22	88	24	94
C5	22	78	20	80	24	86	22	90	24	96	22	106
C6	21	84	22	96	22	96	24	96	24	106	24	106
Average	20	78	20	89	22	94	23	94	24	100	23	105

previously done on the exercise tolerance of the patient with NCA may be misleading because the element of anxiety was not eliminated during such studies. It is a simple point, but one important enough to invalidate any assay of exercise tolerance, if not kept in mind.

Eight patients with NCA and six normal adults were given the test. The results, as shown in Table III, indicate that there was no significant difference in the hemodynamic response of the patient with NCA and that of the normal individual, as judged by pulse and respiration changes during the exercise. The average pulse rate of the patients with NCA increased from 80 to 111 beats per minute at the end of 240 seconds of exercise (an increase of 35.8 per cent), and the average pulse rate of the normal individuals, at the end of the same period, increased from 78 to 105 beats per minute (an increase of 34.6 per cent). The average respiratory rate in both groups was found to remain below 24 per minute during the entire test.

These observations indicate rather conclusively that, if the emotional tachypnea of the patient with NCA is eliminated during an exercise test, no cardiac dysfunction will be observed. Even in the standard test, in which no effort was made previously to acquaint the patient with it, and in which the patient with NCA exhibited persisting tachypnea and tachycardia, evaluation of the cardiac efficiency by measurements of the vital capacity, venous pressure, and circulation time revealed no defect.

It is of interest in this connection that Fraser and Wilson,[16] in commenting on the response of the patient with NCA to exertion, wrote, "These patients appear to differ from healthy men only in that a stimulus such as excitement or emotion produces an unusually large response." Jones and Lewis[17] also observed, "—it is not effort but the situation in which effort may be required and the emotional attitude of the man toward this situation that are often the significant factors." Certainly our own observations are in complete agreement with the views of these men.

E. EXPERIMENTAL OBSERVATIONS CONCERNING THE CARDIOVASCULAR MANIFESTATIONS

1. *Dyspnea.*—Although forty-seven patients with NCA (94 per cent) complained of breathlessness after relatively slight exertion, it was found, as described above, that, when a group of these individuals performed work without any element of concomitant anxiety or emotional tension of any sort, they did not experience dyspnea sooner, or more severely, than the normal person doing the same work. This last observation, together with the lack of signs of cardiac insufficiency in the uncontrolled exercise test described previously, indicated that the respiratory distress of these individuals was not of cardiac origin.

2. *Precordial Pain.*—Forty-four patients with NCA (88 per cent) complained of intermittent precordial pain. This particular symptom, perhaps more than any other, has been responsible for the interest of the cardiologist in neurocirculatory asthenia. Some writers,[18, 19] however, have considered the symptom as imaginary or the result of the patient's preoccupation with his heart. Wood[13] recently has presented very good evidence that precordial pain in many instances may be due to respiratory dysfunction.

In the present studies, it was found that there were actually *two* separate and distinct types of precordial pain which might be experienced by patients with NCA. It was also discovered that they were not due to the same causes. It is probable that needless confusion has existed in the past concerning the nature and cause of the precordial pains occurring in the patient with NCA because of the failure to differentiate these two distinct types.

The most common form of pain (experienced by twenty-four patients with NCA (58 per cent) was a sharp, piercing, transient one that began at the left nipple and penetrated deep into the chest. The intensity was most severe, although the pain rarely persisted over five minutes. The patients described this pain as a sensation of being stabbed, torn, or cut. Eight of the twenty-four patients stated that, either preceding or concomitant with the perception of this pain, they were aware of their hearts beating irregularly. The remaining sixteen patients stated that they were unaware of any irregularity of rhythm, but they did observe that the heart began to beat extraordinarily forcefully, either before or during the attack of precordial pain. Because each of the patients thus insisted that their pain was preceded or accompanied by perceptible changes in either the rhythm or force of cardiac contraction, it was suspected that this type of precordial pain was cardiac in origin.

Accordingly, the susceptible patients were instructed to report for examination immediately at the onset of their precordial pain. Because they were hospitalized patients, thirteen of them were examined almost at the first twinge of pain. It was found on physical and electrocardiographic examination that seven of these patients exhibited some type of transient arrhythmia at the time of the onset of precordial pain (ventricular extrasystoles, 3; auricular paroxysmal tachycardia, 2; wandering auricular pacemaker, 1; and auricular flutter, 1). The remaining six patients were found to have normal electrocardiograms, but the clinical examination revealed that the hearts of all six were pounding very forcefully against the chest wall. Of even greater interest was the observation that ten of these thirteen patients, during their attack of precordial pain, also exhibited cold, wet hands with accentuated tremor, profuse axillary perspiration, and dilatation of the pupils. These extracardiac findings indicated that a nervous discharge had occurred, in addition to the cardiac symptoms and signs. These patients were carefully observed, and it was found that the precordial pain did not persist after the disappearance of the observed arrhythmia and the excessive pounding of the heart against the chest wall.

These findings made it clear that the cause of the sharp, excruciatingly severe, precordial pain was of cardiac origin. It was also obvious that, in the majority of cases, the cardiac changes were but the cardiovascular manifestations of a nervous discharge, strongly suggestive of an excitation of the sympathetic nervous system.

The second type of precordial pain experienced by many patients with NCA was a dull, aching, persistent pain confined, but not sharply limited, to a wide area of the left side of the chest, with the left nipple usually as its center. It was invariably produced by exercise but usually appeared several minutes, to several hours, after the performance of any particular exertion. It was felt by many patients for many hours, contrasting sharply to the sharp type of pain previously described, which rarely lasted over five minutes. It is believed that this is the type of pain described by Wood,[13] who thought it was due to respiratory dysfunction. In data to be published later, sufficient evidence was accumulated to confirm his theory concerning the pathogenesis of this type of precordial pain. It should be mentioned, however, that several patients with NCA in the present series were wont to experience both types of pain at intervals.

3. *Palpitation.*—Forty-seven patients (94 per cent) stated that they had experienced bouts of palpitation. Twenty were examined during hospitalization, at a time when they experienced this sensation. Four were found to have ventricular extrasystoles, and the remaining sixteen patients were observed to have

the pounding of the heart against the chest wall, previously observed in many of the patients with NCA who complained of precordial pain of the sharp, transient variety. As a matter of fact, six of these twenty patients also complained of the sharp precordial pain at the time they experienced palpitation. Fifteen of the twenty patients also showed signs of the nervous discharge.

4. *Changes in the Temperature and Color of the Extremities.*—The cold, cyanotic extremities of the patient with NCA have been described often by various observers.[12, 13] The condition has been thought[13] to be due to arteriolar vasoconstriction. Although forty-five patients with NCA (92 per cent) in the present series exhibited such changes in their extremities at some time during hospitalization, it was not a permanent phenomenon. Rather, it was a transient, episodic condition, associated with the characteristic nervous discharge which has been described as preceding or accompanying the other cardiovascular manifestations of the syndrome. Between these episodes and always during sleep, the skin of the patient with NCA appeared normal, both in temperature and color.

F. THE EFFECTS OF VARIOUS DRUGS ON THE CARDIOVASCULAR SYSTEM

The majority of investigators who have made studies concerning the etiology and pathogenesis of neurocirculatory asthenia have been impressed with the striking similarity of many of its manifestations to those following an excitation of the sympathetic nervous system. Fraser and Wilson[16] were the first observers who stressed the involvement of the autonomic nervous system in the pathogenesis of the syndrome. Kessel and Hyman[20] stressed the presence of autonomic imbalance in the production of neurocirculatory asthenia but did not attempt to correlate their physiologic findings with anatomic elements of the autonomic nervous system. Cannon[21] also pointed out the essential similarity of the syndrome of NCA to the rage or fear reaction occurring in the experimental animal after excitation of the sympathetic nervous system. Despite this admitted involvement of the autonomic nervous system in the pathogenesis of neurocirculatory asthenia, however, the exact locus of its conjunction with, or participation in, the elements making up the entire syndrome remains unknown. For, although the somatic elements of the disease are mediated obviously by the autonomic nervous system, there is little reason to believe that the entire syndrome occurs because of some peripheral disturbance in this latter segment of the nervous organization.

In an effort to investigate the role of the autonomic nervous system in the pathogenesis of this disease, patients with NCA were given various drugs which acted on the peripheral terminations of this system. In addition, they were given two drugs, caffeine and benzedrine, which are known[22] to exert their principal effects on the higher centers of the central nervous system. It was hoped, by such experimentation, that not only the portion of the autonomic system involved in these patients might be determined, but also at what level the function of this system was hyperactive or deranged.

The autonomic effector drugs (epinephrine and physostigmine) were administered to patients with NCA when they exhibited a minimum of cardiovascular manifestations. The anticholinergic drugs (atropine and scopolamine) were given, however, when the patients displayed a marked accentuation of symptoms and signs. Caffeine and benzedrine were given during quiescent periods of the syndrome. These periods (according to the drug employed) were chosen because it would be difficult, for example, to determine the role of epinephrine in a patient with NCA who already was exhibiting maximal signs of the

TABLE IV. THE RESPONSE OF THE PATIENTS WITH NCA TO THE ADMINISTRATION OF VARIOUS DRUGS

CASE	BEFORE ADMINISTRATION								AFTER ADMINISTRATION							
	RESP.	PULSE	TREMOR	PERSPIRATION	SKIN (HANDS)	CARDIAC ARRHYTHMIA	FORCEFUL HEART BEAT	PRECORDIAL PAIN	RESP.	PULSE	TREMOR	PERSPIRATION	SKIN (HANDS)	CARDIAC ARRHYTHMIA	FORCEFUL HEART BEAT	PRECORDIAL PAIN
A. Administration of Epinephrine (0.5 Mg.) by Intramuscular Injection																
a. *Patients With NCA*																
R. G.	20	86	+	+	Warm	Absent	Absent	Absent	20	100	++++	++++	Very cold	Absent	Present	Present
C. N.	18	100	+++	++	Cool	Absent	Absent	Absent	18	112	++++	++++	Very cold	Absent	Present	Present
J. C.	20	84	+	+	Warm	Absent	Absent	Absent	20	108	++++	++++	Very cold	Absent	Present	Absent
H. P.	20	112	++	++	Warm	Absent	Absent	Absent	18	122	++++	++++	Very cold	Absent	Present	Absent
Average	20	96							19	111						
b. *Normal Adult Controls*																
T. T.	18	78	0	0	Warm	Absent	Absent	Absent	20	110	++++	+	Warm	Absent	Present	Present
R. S.	20	82	0	0	Warm	Absent	Absent	Absent	20	108	++++	0	Warm	Absent	Present	Absent
H. Z.	20	80	0	0	Warm	Absent	Absent	Absent	20	105	++++	0	Warm	Absent	Present	Absent
Average	19	80							20	107						
B. Administration of Physostigmine (1 Mg.) by Intramuscular Injection																
Patients With NCA																
H. P.	20	84	+++	+++	Cold	Absent	Absent	Absent	20	84	+++	+++	Warm	Absent	Absent	Absent
J. C.	20	88	+	+	Warm	Absent	Absent	Absent	18	88	+	+	Warm	Absent	Absent	Absent
E. R.	18	84	+++	+++	Warm	Absent	Absent	Absent	20	84	++++	+++	Warm	Absent	Absent	Absent
R. G.	20	88	++	++	Warm	Absent	Absent	Absent	18	86	++	++	Warm	Absent	Absent	Absent
Average	20	86							19	86						
C. Administration of Atropine (1.5 Mg.) by Subcutaneous Injection																
Patients With NCA																
R. S.	24	88	++++	++++	Very cold	Absent	Present	Present	24	120	++++	0	Warm	Absent	Present	Present
R. G.	22	100	++++	++++	Very cold	Absent	Absent	Absent	22	110	++++	0	Warm	Absent	Present	Absent
H. P.	26	96	+++	++++	Very cold	Absent	Present	Present	28	118	++++	0	Warm	Absent	Present	Present
D. W.	24	96	++++	++++	Very cold	Present*	Present	Absent	22	140	++++	0	Warm	Absent	Present	Absent
Average	24	95							24	122						
D. Administration of Scopolamine (0.6 Mg.) by Subcutaneous Injection																
Patients With NCA																
E. R.	24	94	+++	++++	Very cold	Absent	Present	Absent	24	92	++++	++++	Warm	Absent	Present	Absent
H. P.	22	88	++++	++++	Very cold	Absent	Present	Present	22	94	++++	++++	Warm	Absent	Present	Present
E. K.	23	96	++++	++++	Very cold	Absent	Present	Absent	24	98	++++	++++	Warm	Absent	Present	Absent
Average	23	93							23	95						

E. Administration of Caffeine (500 Mg.) by Oral Ingestion

a. *Patients With NCA*

D. W.	20	76	++	+	Warm	Absent	Absent	Absent	30	96	++++	++++	Very cold	Present*	Present	Present
D. C.	20	88	++	+	Warm	Absent	Absent	Absent	24	84	++++	++++	Very cold	Absent	Present	Absent
W. B.	24	86	++	+	Warm	Absent	Absent	Absent	30	112	++++	++++	Very cold	Absent	Present	Absent
E. R.	16	74	+++	+++	Cool	Absent	Absent	Absent	28	90	++++	++++	Very cold	Absent	Present	Present
H. P.	20	86	+	+	Warm	Absent	Absent	Absent	24	96	++++	++++	Very cold	Absent	Present	Absent
M. N.	20	84	+	+	Warm	Absent	Absent	Absent	22	108	++++	++++	Very cold	Absent	Present	Present
Average	20	82							26	98						

b. *Normal Adult Controls*

E. H.	18	88	0	0	Warm	Absent	Absent	Absent	16	72	0	0	Warm	Absent	Absent	Absent
R. C.	20	60	0	0	Warm	Absent	Absent	Absent	20	60	0	0	Warm	Absent	Absent	Absent
T. S.	18	88	0	0	Warm	Absent	Absent	Absent	20	78	0	0	Warm	Absent	Absent	Absent
Average	19	79							19	70						

F. Administration of Benzedrine (10 Mg.) by Oral Ingestion

a. *Patients With NCA*

R. S.	20	85	+	++	Warm	Absent	Absent	Absent	20	108	+++	+++	Very cold	Present*	Present	Present
H. E.	20	64	++	+	Warm	Absent	Absent	Absent	32	98	++++	+++	Very cold	Absent	Present	Present
D. C.	26	76	+	++	Warm	Absent	Absent	Absent	28	79	++++	++++	Very cold	Absent	Present	Present
H. P.	20	91	++	+	Warm	Absent	Absent	Absent	32	109	++++	+++	Very cold	Absent	Present	Absent
F. M.	—	90	+	+	Warm	Absent	Absent	Absent	—	95	++++	++++	Very cold	Absent	Present	Absent
R. G.	20	89	++	+	Warm	Absent	Absent	Absent	28	106	++++	++++	Very cold	Present*	Present	Present
Average	21	83							28	99						

b. *Normal Adult Controls*

E. Y.	18	87	0	+	Warm	Absent	Absent	Absent	18	86	0	+	Warm	Absent	Absent	Absent
W. R.	20	60	0	0	Warm	Absent	Absent	Absent	20	69	0	0	Warm	Absent	Absent	Absent
W. W.	20	75	0	+	Warm	Absent	Absent	Absent	20	76	0	+	Warm	Absent	Absent	Absent
Average	19	74							19	77						

*Ventricular extrasystoles.

syndrome before medication. Likewise, the use of anticholinergic drugs during a quiescent period of the syndrome would be without value in assessing the role of the parasympathetic nervous system in the production of neurocirculatory asthenia. These precautions are extremely important in dealing with a disease so episodic in character, and some of the conflicting views concerning the results following the administration of various autonomic drugs may have arisen because of the failure to ascertain the exact status of the patient before these drugs were given.

1. *The Administration of Epinephrine.*—Inspection of Tables IV, *A*, shows clearly that the intramuscular injection of epinephrine produced no greater changes in the patient with NCA, for the most part, than in the normal individual. In both groups, the injection was followed by increase in pulse rate and force of cardiac contraction. Increase in the force of the heart beat by this procedure produced the sharp type of precordial pain in two patients with NCA and in one normal individual. The only real difference observed was the occurrence of cold, wet hands in the patients with NCA, which was observed to occur only after they had begun to feel anxiety about the forceful contraction of the heart making it doubtful that the epinephrine directly evoked the changes in the peripheral blood vessels and sweat glands. The pre-existing tremor of the patients with NCA was seen to increase, but all normal adults also exhibited a tremor following the injection of the drug.

2. *The Administration of Physostigmine.*—The intramuscular injection of physostigmine into four patients with NCA (Table IV, *B*) during a relatively quiescent period of the syndrome, evoked no essential change in the pulse rate, the degree of perspiration, or in the color and temperature of the upper extremities. The tremor did not increase in any patient. It appeared, then, that the cardiovascular apparatus of the patient with NCA was not hypersusceptible to this cholinergic drug. By inference, it would seem that these patients were also not unduly sensitive to parasympathetic discharge.

3. *The Administration of Atropine.*—The subcutaneous injection of atropine into four patients with NCA (Table IV, *C*) during exacerbations of symptoms and signs, had no effect in inhibiting the increase in respiratory rate or tremor of the extremities. The cardiac rate was increased in even greater degree. Atropine, however, did abolish the excessive perspiration and the coldness of the hands. It should be mentioned in this connection that, although perspiration is a function of the sympathetic nervous system, the effector drug actually causing the increased perspiration has been found to be acetyl choline.[22] This makes it clear that the abolition of excessive perspiration by the injection of atropine does not indicate that a previously increased parasympathetic discharge was present in these patients.

4. *The Administration of Scopolamine.*—The subcutaneous injection of scopolamine into three patients with NCA, during exacerbations of the disease, was followed (Table IV, *D*) by the disappearance of previously cold extremities. The other manifestations of the syndrome were unchanged after injection.

5. *The Administration of Caffeine.*—When caffeine citrate was given orally to six patients with NCA during quiescent periods of their illness and to three normal young adults (for control purposes), a marked effect was observed only in the patients with NCA (Table IV, *E*). Five of the six patients exhibited an increase in the pulse rate (average increase: 16 beats per minute). Each of the six patients showed an increase in respiratory rate (average increase: 6 respirations per minute). The tremor also became accentuated, and the hands

of all patients with NCA became wet and cold. The impact of the heart against the chest wall was observed to increase in intensity in all patients and three patients with NCA complained of the sharp, transient type of precordial pain. One patient had a transient arrhythmia, due to the onset of ventricular extrasystoles. As mentioned, the normal individuals showed no significant change in pulse or respiratory rate, in rhythm, or force of cardiac contraction, or in degree of perspiration.

6. *The Administration of Benzedrine.*—Benzedrine sulphate produced dramatic changes when given to six patients with NCA, but there were no perceptible changes in the normal individuals to whom it was given. Thus (Table IV, *F*) the average pulse rate of the six patients with NCA increased from 83 to 99 beats per minute, and the respiratory rate accelerated from 21 to 28 breaths per minute. Each of the six patients complained of palpitation, and four experienced the sharp, transient type of precordial pain. The hearts of all patients with NCA were observed to contract much more forcefully after the administration of benzedrine and, in two patients, ventricular extrasystoles followed the ingestion of the drug. The hands of all patients, regardless of their state prior to the administration of benzedrine, became cold and wet. Two patients also exhibited purplish mottling of the skin of their extremities. Axillary perspiration increased in all patients following the ingestion of benzedrine.

DISCUSSION

From the preceding observations, it must be assumed that the heart of the patient with NCA is basically sound both in structure and function. If, occasionally at rest, and always during effort associated with emotional activity, this type of patient experienced dyspnea, palpitation, and precordial pain, and exhibited tachypnea, forceful beating of the heart, arrhythmia, and coldness of the extremities, there was sufficient evidence also that these cardiovascular symptoms and signs were preceded or accompanied by a nervous discharge which seemed to bear a causal relationship to the onset of the cardiovascular dysfunction observed.

Thus, it was found that the real mechanism responsible for the occurrence of not only the cardiovascular manifestations, but also of the other somatic manifestations of the disease, was the episodic, frequently spontaneous occurrence of this nervous discharge. The cardiac and extracardiac characteristics, the close relationship to emotion, and the experimental simulation of these signs and symptoms in the normal individual by the administration of excessive amounts of epinephrine, indicated that the nervous discharge was sympathetic in nature. The inability to reproduce an exacerbation of the syndrome in the patient with NCA by the administration of cholinergic drugs, further confirmed this probability.

However, the peripheral portion of the sympathetic nervous system in the patient with NCA was not found to be unduly sensitive to epinephrine, which suggested that the abnormality in this portion of the autonomic nervous system was not a peripheral one. The production of the same type of nervous activity by emotional activity and by the administration of caffeine and benzedrine (drugs having their chief effect on the higher centers of the central nervous system[22]) indicated that there was stimulation of the sympathetic nervous system at its central point. Since the hypothalamus has been described[23] as the locus of this point, it would appear that this portion of the brain in the patient with NCA was unduly responsive to emotional activity or to the administration of the

aforementioned drugs. The presence of fever in many patients with NCA[1] together with other symptoms and signs previously described[2] further suggests hypothalamic dysfunction in this illness.

This incrimination of the hypothalamic area of the brain in the pathogenesis of neurocirculatory asthenia is not to be construed as a belief that hypothalamic dysfunction is responsible for the genesis and pathogenesis of the entire syndrome. It is believed, however, that the evidence is strong for the assumption that this portion of the brain is responsible for the emergence of the somatic manifestations seen in this disease. It should be pointed out, in this connection, that neurocirculatory asthenia is intimately associated with emotion and the latter, in turn, has close relationship with the hypothalamus.

CONCLUSIONS

1. The cardiovascular manifestations of neurocirculatory asthenia were studied in fifty patients.

2. The heart of the patient with NCA was found to be normal in size, structure, and function at rest and also during effort if unaccompanied by emotional activity.

3. Changes in the rate, rhythm, and force of cardiac contractions were observed in the patient with NCA to be preceded by, or associated with, excitation of the sympathetic nervous system. This phenomenon was observed during a natural exacerbation of the syndrome or following the administration of suitable drugs.

4. Evidence was obtained which suggested that the excitation of the sympathetic nervous system, preceding or associated in a causal fashion with the cardiovascular manifestations of neurocirculatory asthenia, resulted from hypothalamic discharge.

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PULMONARY STENOSIS WITH INTACT INTERVENTRICULAR SEPTUM

Report of Eleven Cases

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STENOSIS of the pulmonary valve is encountered quite frequently in infants and young adults with congenital heart disease and is usually associated with the other three components of the tetralogy of Fallot, namely, interventricular septal defect, transposition of the aorta, and hypertrophy of the right ventricle. In Maude Abbott's[1, 2] series of 1,000 cases of congenital heart disease, pulmonary stenosis was associated with an interventricular septal defect in eighty-five cases and, in thirty-four of these, the foramen ovale was also patent. However, in twenty-five other cases, she found pulmonary stenosis associated with an intact interventricular septum; sixteen of these demonstrated patency of the foramen ovale. Pulmonary atresia is not considered, because it is of little clinical significance and is usually incompatible with life. Thus, according to Abbott's findings, at least two patients in seven with pulmonary stenosis will not have a ventricular septal defect, but this has not been adequately recognized in general or, even as a rule, by cardiologists, themselves.

Interest in pulmonary stenosis with intact interventricular septum was stimulated recently by the observation of three such cases. The clinical diagnosis was in error in two of the cases while, in the third case, a correct diagnosis was made. Search of the autopsy protocols of the Boston City Hospital, Massachusetts General Hospital, and Children's Hospital revealed a total of eleven cases in which pulmonary stenosis was not associated with a ventricular septal defect. An analysis of these cases with the pertinent physical and anatomic findings, together with the available electrocardiograms and roentgenograms, form the basis for this report. A few remarks concerning etiology likewise seem appropriate.

Case 1 (R. J., 39805).—The patient was a 12-year-old boy, first seen in the Outpatient Department of the Massachusetts General Hospital in April, 1937, when he was referred from the Children's Hospital. In March, 1929, at the age of 4 years and 4 months, he had been examined in the Children's Hospital, at which time some cyanosis of the fingers and lips, but no clubbing of the fingers or toes, was observed. The blood pressure at that time was 110/60.

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The hemoglobin was 55 per cent and the red blood cell count was 4.08 million per cubic millimeter. His mother had noted some cyanosis of the face and internal strabismus since the age of 3 years. In October, 1930, a definite bulge was noted in the precordial region, and he was observed to be somewhat dyspneic on exertion. In April, 1932, his hemoglobin was 95 per cent; his red blood cell count was 5.7 million and his white blood cell count was 13,000 per cubic millimeter. The diagnosis of the tetralogy of Fallot was thought to explain best the congenital anomaly of the heart. In May, 1936, an electrocardiogram was taken which revealed evidence of marked right ventricular enlargement. He had been treated at the Massachusetts Eye and Ear Infirmary, from the time he was 8 until he was 12 years of age, with optical glasses for internal strabismus, with good results.

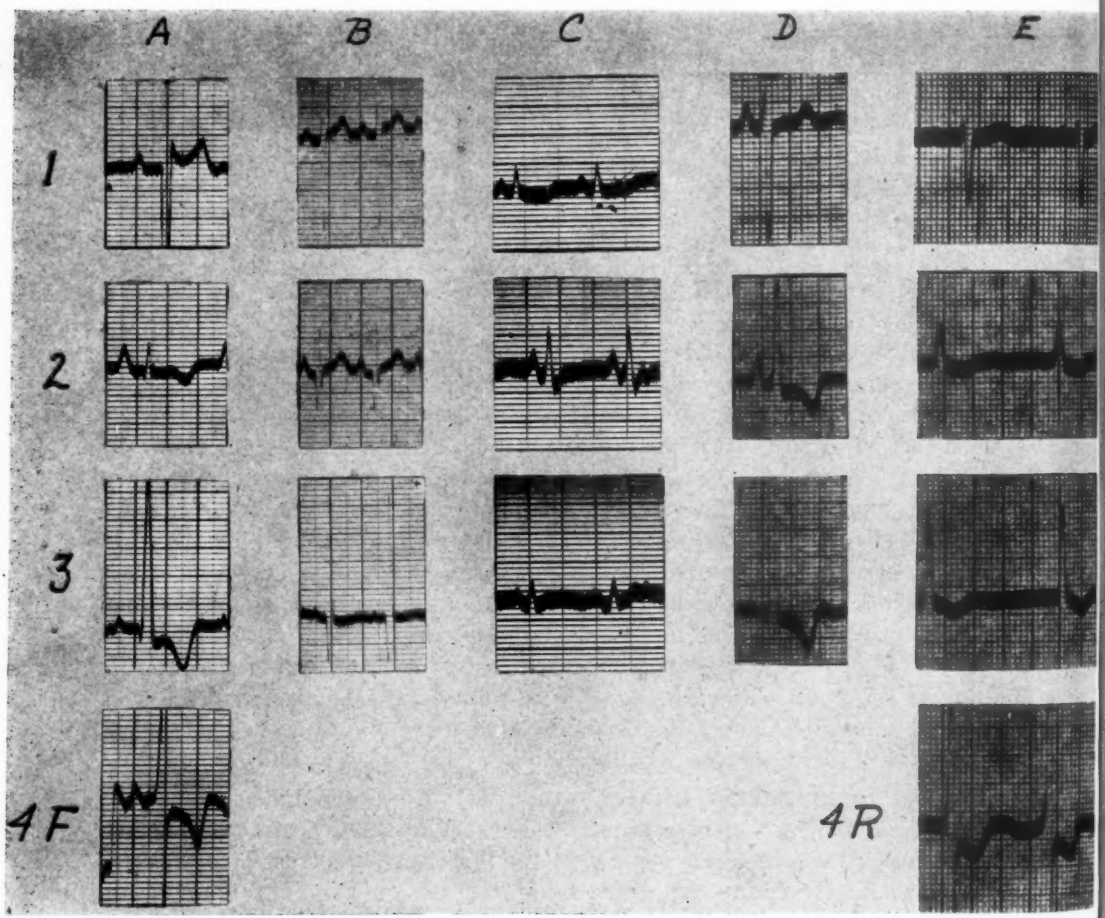
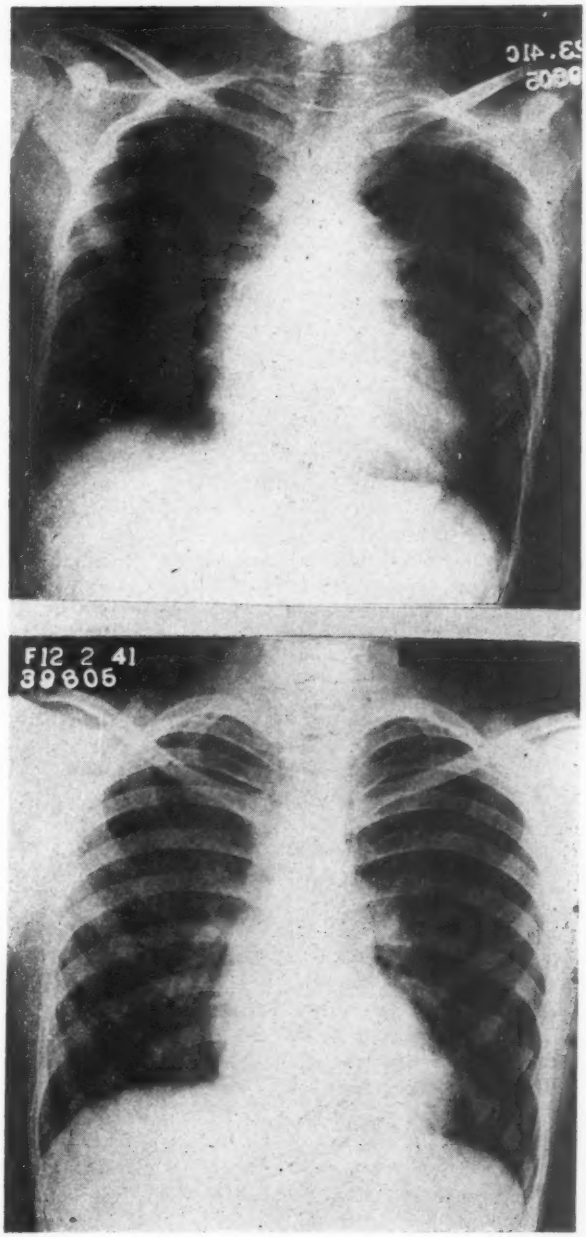


Fig. 1.—The electrocardiograms of four patients with pulmonary stenosis. *A*, Case 1. The tracings made in 1937 and 1941 were essentially the same. The high degree of right axis deviation, inversion of T waves in Leads II, III, and IVF, and prominent P waves should be noted. *B*, Case 2. The high degree of right axis deviation is apparent; the Q waves in Leads II and III are prominent and measure 6 millimeters. The T wave in Lead III is isoelectric. *C*, Case 4. There is no evidence of right ventricular strain in this tracing. It is, however, similar to the changes seen in some cases of pellagra.[13] The short P-R interval and low T waves should be noted; the T wave in Lead I is slightly inverted. *D* (April 8, 1937) and *E* (May 6, 1941), Case 9. The tracing is quite similar to that of Case 1. Auricular fibrillation developed shortly before death (*E*).

The physical examination revealed a small, intelligent, blond male, exhibiting mild cyanosis of the lips, cheeks, and fingers, with slight clubbing of the fingers. The left border of dullness was 8 cm. to the left of the midsternal line in the fifth intercostal space, 1 cm. beyond the midclavicular line. There was a moderately loud, but not intense, rough systolic murmur in the pulmonic area, with a very slight thrill; the murmur was heard also, less loudly, at the apex. The second sound at the pulmonic area was of good quality and was equal in intensity to the aortic second sound. The blood pressure was 95/70. The electrocardiogram revealed normal rhythm, with a rate of 90 per minute, with marked right axis

deviation and prominent P waves. The T waves in Leads II, III, and IVF were inverted (Fig. 1, *A*). The x-ray film of the chest demonstrated enlargement of the heart, most striking at the base of the heart, with some prominence of the pulmonary artery (Fig. 2, *A*). Because of the appearance of the heart by x-ray examination, which showed a prominent pulmonary artery, the diagnosis of Eisenmenger's complex (ventricular septal defect, dextroposition of the aorta, dilated pulmonary artery, and a large right ventricle) was made.

In October, 1939, the cyanosis and clubbing of the fingers were little changed from that observed previously. However, by July, 1941, the cyanosis had become greater, though still moderate in degree, there was noted marked prominence of the precordium, and the pulmonary second sound was thought to be less loud than it normally is for a person his age.



A.

B.

Fig. 2.—Roentgenograms in Case 1. *A*, which is not a teleroentgenogram, was made July 23, 1941. *B* is a teleroentgenogram made Dec. 2, 1941. Each film demonstrates slight cardiac enlargement and prominence of the pulmonary artery. There is also a partial left pneumothorax present in the lower film. Because of the prominent pulmonary artery, the diagnosis of Eisenmenger's complex was made.

In December, 1941, the patient was brought to the hospital because of severe pain in the left side of the chest, followed by nausea and vomiting. The x-ray film of the chest (Fig. 2, *B*) demonstrated a partial left pneumothorax. The pain subsided after one day, and he returned home. In April, 1942, the cyanosis and clubbing were described as marked. The patient was clearly undersized for his age, weighing 84 pounds and measuring 5 feet, 4 inches in height. A slight venous pulse was noted in the neck at this time. In August, 1942, the hemoglobin was 22.7 Gm., and the red blood cell count was 8.65 million per cubic millimeter. In September, 1942, he was mostly confined to bed, after several episodes of fainting, which were followed by moderate dyspnea. He was finally admitted to another hospital where he died, with moderate respiratory failure, in November, 1942. The clinical diagnosis was congenital heart disease, Eisenmenger's complex.

Autopsy Findings.—Anatomic diagnosis: (1) Congenital cardiac anomaly: (a) fusion of the cusps of the pulmonic valve, with marked stenosis; and (b) insufficiency and patency of the foramen ovale. (2) Cardiac hypertrophy, the heart weight being 380 grams. (3) Platelet thrombus on the pulmonic valve. (4) Chronic passive congestion of liver, spleen, pancreas, and kidneys.

The heart was globular in shape; the right side appeared larger than the left, and the apex was formed by the right ventricle. The foramen ovale was patent and admitted a 1-cm. probe with ease (Fig. 3, *A*). The right ventricular wall measured 1.5 cm. in thickness, as compared with 1.3 cm. for the wall of the left ventricle. The valves were normal except for the pulmonary valve, whose cusps were completely fused (Fig. 3, *B*). The orifice was oval and measured 6 mm. in diameter. Attached loosely, at the right side of the opening, was an irregularly shaped friable thrombus, 6 mm. in length. The pulmonary artery was moderately dilated, the ductus arteriosus was closed, and the interventricular septum was closed.

Microscopic examination demonstrated that the small thrombus on the pulmonary valve consisted largely of platelets and fibrin. There was no evidence of an increase in connective tissue in the liver, but there was marked congestion of the blood vessels.

CASE 2 (L. M., 401880).—A 3-month-old female infant entered the Massachusetts General Hospital on May 5, 1943, because of convulsions. Two weeks prior to entry, while lying in the crib crying, the patient was observed to stiffen suddenly for a moment and then lie completely limp, breathing stertorously. This occurred again the day of admission. The patient was the victim of an arduous delivery which lasted four days and was finally terminated by rotation and extraction with forceps. The patient had had a face presentation and the mother, a contracted pelvis. The infant weighed 7½ pounds at birth, and was said to be cyanotic; resuscitation was accomplished with difficulty. However, since birth, no cyanosis, dyspnea, or cough had been noted.

The physical examination revealed an infant who showed little interest in her surroundings. The eyes were directed downward to the left and the upper extremities were trembling. The pupils were equal and reacted to light. The heart was enlarged to the left; the left border of dullness measured 2 cm. beyond the midclavicular line. A loud, harsh systolic murmur was heard over the precordium, maximal at the pulmonary area, and was also heard over the back. The second heart sound in the pulmonary area was present, but diminished in intensity. The blood pressure was 90/40. No cyanosis or clubbing was present. The abdomen was normal and the deep tendon reflexes were present. Moderate talipes varus was present bilaterally.

The laboratory examination revealed a hemoglobin of 14.2 Gm. The erythrocytes numbered 4.40 million and the leucocytes 13,400 per cubic millimeter. The blood smear and differential count were not remarkable. The urinalysis was normal. A tourniquet test was negative and the bleeding time was four minutes. The prothrombin time was 17 seconds (with a normal of 18 seconds). An x-ray film of the skull revealed evidence of moderate bulging of the fontanels. The x-ray examination of the chest revealed a heart which was moderately enlarged, as illustrated in Fig. 4. The electrocardiogram (Fig. 1, *B*) showed normal rhythm, at a rate of 150 to 160 per minute. The P-R interval measured 0.12 seconds, the Q_2 and Q_3 deflections measured 6 mm., and there was marked right axis deviation.

A cerebral "ventricular" tap was attempted on the day of entry; it revealed grossly bloody fluid with an erythrocyte count of 560,000 per cubic millimeter and xanthochromic supernatant fluid after centrifuging. The total protein of the supernatant fluid was 2,160 mg. per cent. Another "ventricular" tap, attempted the following day, yielded similar results. A lumbar puncture on May 8, 1943, revealed grossly bloody fluid with xanthochromic supernatant fluid. However, the fluid removed by lumbar puncture became less bloody and, by May 18, the fluid contained only 20 erythrocytes per cubic millimeter, and the total protein was

22 mg. per cent. The pressure, however, was elevated, ranging from 250 to 400 cm. of water, as the initial pressure, and this elevation continued, in spite of lumbar punctures every one or two days. On June 2, a combined lumbar and "ventricular" tap revealed grossly bloody fluid from the "ventricles" with xanthochromic fluid, while the fluid from the spinal puncture was clear. Dye, injected into the "ventricular" needle, failed to appear in the lumbar region. On July 8, bilateral frontal exploration with Burr holes was done, and bilateral subdural fluid was obtained. The condition of the patient changed little and, on July 14, an attempt was made to remove the subdural sacs, but the patient stopped breathing during the operation.

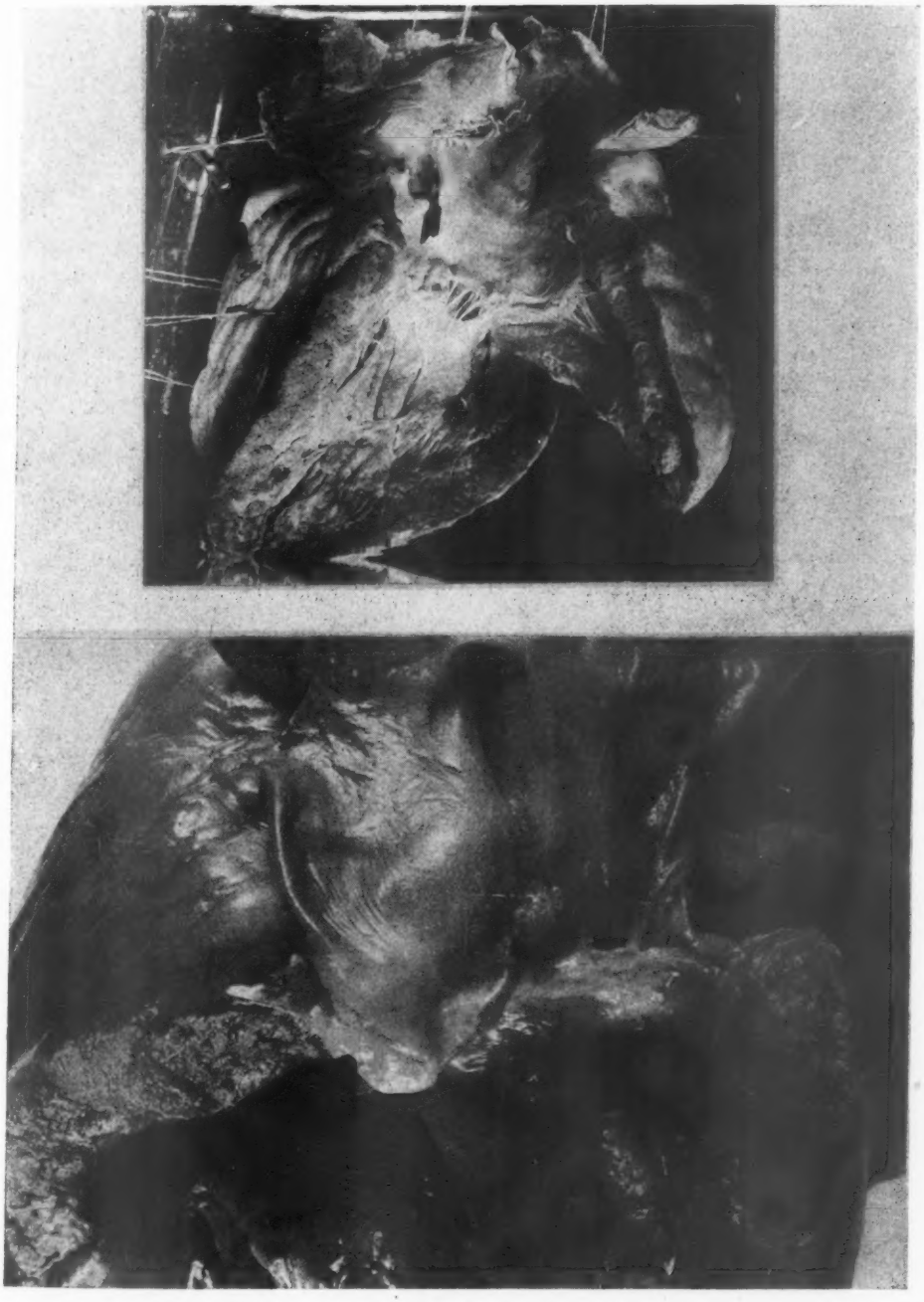


Fig. 3.—The heart in Case 1. *A*, The right auricle and ventricle are open and the marked hypertrophy of the right ventricle can be seen. The arrow on the left is placed through the patent foramen ovale, while the arrow on the right is placed through the stenotic pulmonary valve. *B*, A close view of the pulmonary valve, demonstrating the fusion and scarring of the pulmonary valve leaflets and a small vegetation attached to the superior surface. Moderate dilatation of the pulmonary artery, distal to the valve, is also evident.

The clinical diagnoses were as follows: (1) subdural hematoma, bilateral; (2) hydrocephalus; (3) cyst of the corpus callosum; (4) talipes varus; and (5) interventricular septal defect.

Autopsy Findings.—Anatomic diagnosis: (1) bilateral subdural hematoma; (2) congenital heart disease with pulmonary stenosis; and (3) previous operations: (a) bilateral frontoparietal exploratory trephining, and (b) right craniotomy, with removal of hematoma and its membrane.

The heart weighed 50 grams, and there was prominence of the right ventricle. The right ventricular wall measured 9 mm., and the left ventricular wall measured 7 mm. in thickness. The columnae carneae were hypertrophied in the right ventricle. The foramen ovale was patent, but not dilated, and there was no evidence that any appreciable amount of blood had

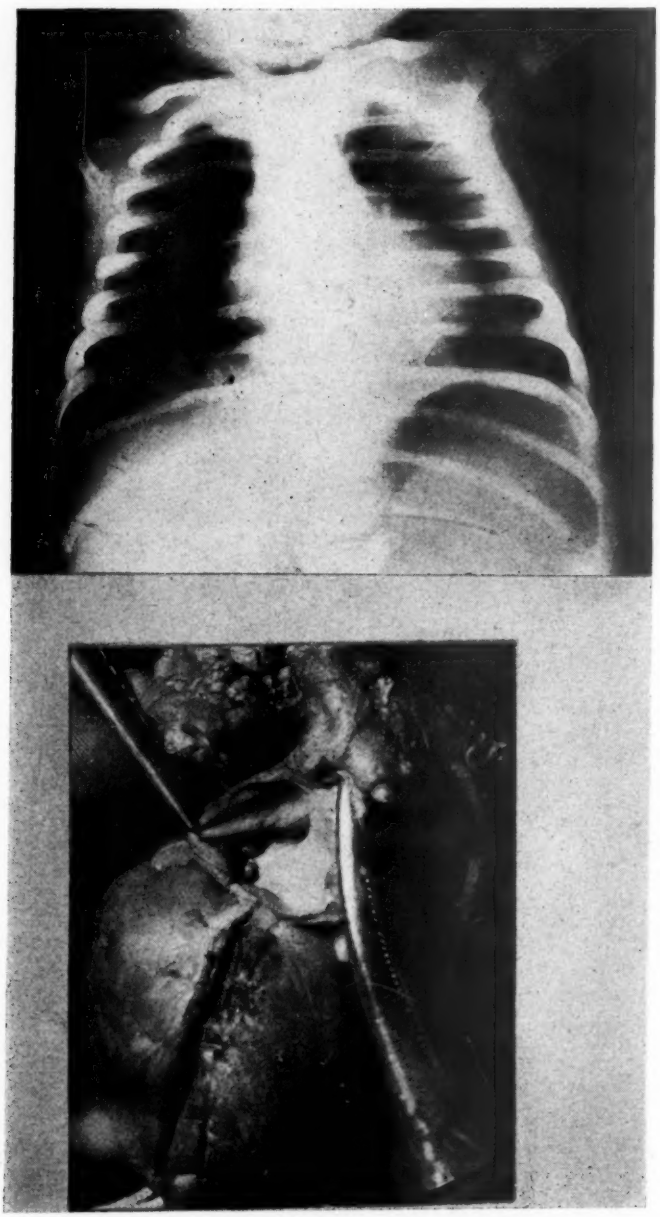


Fig. 4.—*A*, The roentgenogram in Case 2, taken May 7, 1943. This is not a teleroentgenogram. The heart is, however, moderately enlarged. *B*, The heart in Case 2, showing the prominence of the right ventricle. The thickened cusps of the pulmonary valve can be seen and the marked stenosis of the valve is apparent. The pulmonary artery, distal to the valve, is moderately dilated and the wall is delicate and thin.

been able to pass through it. The valve leaflets of the pulmonary artery were distinct and separate, but there was marked stenosis of the annulus, which measured 1 cm. in circumference. The valve leaflets were thickened, and the pulmonary artery in the first portion was thin and somewhat dilated (Fig. 4, *B*). The aortic valve was normal and measured 2.5 cm. in circumference, and the tricuspid and mitral valves were normal. Each measured 3.5 centimeters. The interventricular septum was intact and the ductus arteriosus was closed. The liver was normal. Sections for microscopic examination of the pulmonary valve were not made.

CASE 3 (G. F., A-303).—A 22-year-old, married woman entered the Massachusetts General Hospital April 20, 1898, complaining of dyspnea and cough of four months' duration, which had become progressively worse for two weeks. She had had two children without difficulty; the younger was 2 years of age. "Heart trouble" had been present for several years, but no history was given of her condition at birth. No history of rheumatic fever was obtained.

The physical examination revealed marked cyanosis of lips, cheeks, ears, and nails, and moderate cyanosis of the skin over the body. The lungs were normal. The heart was enlarged, both to the left and right by percussion, with the left border 1 cm. to the left of the nipple line. No thrill was palpated but a harsh systolic murmur was well heard over the precordial area. In the third and fourth intercostal spaces, to the left of the sternum, the systolic murmur had a higher pitch. The heart sounds at the base were of poor quality and the pulmonary second sound was accentuated. The pulse was regular, with a rate of 90 per minute, and the respiratory rate was 30. The liver extended 5 fingerbreadths below the right costal border and moderate pitting edema was present over the lower extremities. The urine examination demonstrated a small amount of albumin. The edema diminished while on bed rest, and she was discharged, improved, on May 2, 1898.

On June 22, 1898, the patient re-entered the hospital because of increasing edema and dyspnea and right hemiparesis for two weeks. Physical examination was little changed except for a hemiparesis of moderate degree. She was again discharged on July 7, 1898, considerably improved after having received small doses of the tincture of digitalis.

On July 29, 1898, she returned to the hospital because of increasing dyspnea and cyanosis, being unable to lie flat in bed. A sister was said to have died of heart trouble, two weeks before the patient's re-entry. Physical examination revealed intense cyanosis and moderate orthopnea. The lungs were normal. The heart sounds were less loud, and the pulse rapid and regular with a rate of 120 per minute. The right hemiparesis was less marked, and there was little dependent edema. Moderate tenderness was elicited over the liver. The cyanosis increased and, by July 31, the cyanosis was extreme, yet the lungs were negative to examination. Later the same day, she died. A clinical diagnosis was made of chronic endocarditis with mitral regurgitation and, possibly, congenital heart disease.

Autopsy Findings.—Anatomic diagnosis: (1) stenosis of pulmonary valve of heart, congenital; (2) hypertrophy and dilatation of right auricle and ventricle; (3) small, patent foramen ovale; (4) mural thrombosis of right auricular appendage, right ventricle, and left ventricle; and (5) chronic passive congestion of the liver.

No pleural fluid was found, and the lungs were not unusually heavy. The heart weighed 630 grams. The right auricle was unusually large, the cavity being about the size of a lemon. The right ventricle was prominent, and the cavity was large. The right ventricular wall measured 12 mm. in thickness. The cusps of the pulmonary valve were joined to each other and were connected with the wall of the pulmonary valve by three small, smooth bands of tissue; the appearance was produced of four small cusps, each about one-half the size of the aortic cusps. The foramen ovale was open and measured about 5 by 5 millimeters. The left ventricle was slightly dilated, but otherwise was normal. The remaining valves were normal. The interventricular septum was intact. The liver was not remarkable, except for chronic passive congestion.

CASE 4 (E. A. P. E., A5203).—A 43-year-old actress entered the Massachusetts General Hospital July 8, 1927, complaining of progressive weakness and weight loss over a period of ten months. Since early childhood, the patient had had much indigestion, with intermittent episodes of upper abdominal pain, lasting several days and occurring two or three times a year. Her appetite had always been fastidious; she had an aversion to meat and seldom ate red meat but occasionally ate fish. During the ten months preceding entry, she had also noted generalized edema and ascites, with some hyperesthesia of the skin and frequent drenching

night sweats. For the same period of time, she had had four to six small watery stools each day, a sore mouth, and dark, red, circumscribed areas over the dorsum of each hand, varying from week to week. For an indefinite period, some loss of memory and mental confusion had been present. There had been no history of dyspnea, cyanosis, or heart disease.

The physical examination revealed a fairly well-nourished, middle-aged woman with generalized anasarca and cutaneous hyperesthesia. The face was edematous, and the skin and lips were moderately cyanotic. The tongue was red and raw and, over the dorsum of each hand, was a dusky red discoloration with a good deal of crustation. The lungs were normal to auscultation. The heart size was difficult to discern by physical examination. The heart sounds were of good quality and a loud systolic murmur was heard in the second and third intercostal spaces, to the left of the sternum. It was well heard over the posterior chest. A short rough distolic murmur was also faintly audible at the pulmonic area. The pulmonary second sound was of good quality and equal to the aortic second sound. The blood pressure was not measured because of the edema and hyperesthesia of the skin. The pulse rate on entry was 100 and varied from 90 to 120 during the hospital stay. The abdomen was distended and a large, irregular mass was palpable in the upper abdomen in the region of the liver. The lower extremities were quite edematous and the deep tendon reflexes were hyperactive.

The laboratory data revealed a hemoglobin of 85 per cent; the red blood cell count was 5 million and white blood cell count was 12,000 to 18,000 per cubic millimeter, with a normal differential count. The urine examination was normal. Blood chemical studies revealed the following: nonprotein nitrogen, 42 mg. per cent; sugar, 117 mg. per cent; and total serum protein, 6.1 Gm. per cent. A "portable" chest x-ray film revealed a slight prominence of the pulmonary conus but no appreciable enlargement of the heart. An electrocardiogram demonstrated a sinus tachycardia of 120 per minute, slightly depressed S-T_1 segment, flat T waves in Leads I and II, a low T_2 wave, and a P-R interval of about 0.10 second (Fig. 1, *C*).

During the first ten days, the patient received 0.2 Gm. of digitalis every day, without any appreciable improvement. A high-vitamin, nutritious diet was likewise given without any striking benefit. She was, at times, confused while in the hospital but, at other times, was very lucid. An abdominal paracentesis on July 21 yielded 2 liters of lemon yellow, cloudy fluid, with a specific gravity of 1.010. The diarrhea continued, her mental confusion increased, and generalized edema became worse. She developed a high fever and died on July 31, 1927. The clinical diagnoses were as follows: (1) pellagra; (2) abdominal neoplasm; (3) slight pulmonic stenosis, probably congenital; and (4) generalized anasarca.

Autopsy Findings.—Anatomic diagnoses: (1) carcinoma of the ileum with metastases to the liver, ovaries, and thoracic duct; (2) pellagra; (3) congenital heart disease, with pulmonary stenosis; (4) chronic passive congestion of the liver; and (5) ascites.

About 500 c.c. of slightly cloudy, blood-tinged fluid were present in each pleural cavity. There was moderate congestion of each lung, particularly in the lower lobes. The heart weighed 260 grams. The right ventricular wall measured 6 mm. in thickness and was definitely hypertrophied. The left ventricular wall measured 1.9 cm. in thickness, and the columnae carneae and the ventricular cavity were normal. The pulmonary valve measured 4.5 cm. in thickness and was moderately stenotic. The valve edges were thicker and more fibrous than normal. The surface of the valve was smooth. The mitral, tricuspid, and aortic valves were normal. There was marked chronic passive congestion of the liver with some atrophy of the liver-cell cords.

CASE 5 (P. S., A-2753).—A 30-year-old salesman entered the Massachusetts General Hospital Dec. 22, 1910, complaining of chills, fever, headache, and cough for four days. The family history was irrelevant. Since infancy he had noted dyspnea on exertion, but this had never been sufficient to confine him to bed. He had also noted a bluish discoloration of the skin at times since infancy, which was especially noticeable whenever he became chilly. At the age of 23 years, the patient had rheumatism. In August, 1910, he was seen in the Medical Outpatient Department where a diagnosis of mitral regurgitation was made. The hands were noted to be cold and blue at this time. During the six months before entry to the hospital, the dyspnea on exertion had increased. He had not been in bed before entry to the hospital.

The physical examination, on entry, revealed a fairly well-developed man who coughed frequently. The extremities and ears were cold and deeply cyanotic. The cyanosis decreased appreciably after he became warmer. There was no clubbing of the fingers or toes. The heart was found to be enlarged, with a diffuse impulse over the precordium, palpable as far as 3 cm. to the left of the nipple line in the fifth intercostal space. The left border of dullness was 2 cm. to the left of the nipple line in the sixth intercostal space. There was a precordial systolic murmur which was best heard in the third and fourth intercostal spaces to the

left of the sternum and was transmitted to the neck, left axilla, and back. The pulmonary second sound was fairly forceful, but indistinct, soft, and rather obscured by the systolic murmur. The aortic second sound was not heard. The systolic blood pressure was 105 mm. Hg, systolic, and the diastolic was indistinct. The pulse was regular and full, with a rate of 100 per minute. The chest examination revealed only a few moist râles at the base of each lung and in the axilla, without bronchial breath sounds. The abdomen was negative.

The laboratory studies demonstrated a hemoglobin of 90 per cent; the white blood cell count was 17,700 and the red blood cell count was 5.8 million per cubic centimeter. The urine contained 3 plus albumin and a moderate number of granular casts. The sputum was like prune juice and contained pneumococci. The temperature was 104° F.

The day after entry, bronchial breath sounds and egophony were noted over the right upper and middle lobes of the lung, with numerous moist râles. The cyanosis increased, and his fever remained at 104° F. He became delirious and died two days after entry. The clinical diagnosis was lobar pneumonia, chronic mitral endocarditis, and a question of congenital heart disease.

Autopsy Findings.—Anatomic diagnoses: (1) lobar pneumonia, right lung; (2) chronic pleuritis, right; (3) stenosis of the pulmonary valve, congenital; (4) fibrous endocarditis of the tricuspid valve, with slight stenosis; (5) hypertrophy and dilatation of the heart; (6) defective closure of the foramen ovale; (7) chronic passive congestion.

The heart weighed 430 grams. The right ventricular wall was greatly thickened and measured 10 to 15 mm. in thickness. The right auricular wall was also thickened and the columnae carneae were markedly thickened. The right auricular cavity was large. The pulmonary valve was about 5.5 mm. in diameter and about 17 mm. in circumference. The orifice of this valve admitted the passage of an ordinary lead pencil. There were three cusps which showed a moderate amount of diffuse fibrous thickening, with fusion of the contiguous cusp margins. The pulmonary artery and its branches were small, but the branches in the lung were of good size. The tricuspid valve measured 10 cm. in circumference, and the leaflets showed a moderate amount of diffuse fibrous thickening which, in places, was quite nodular. The mitral valve circumference was 10 cm., and the cusps were slightly thicker than usual, but the valve was otherwise normal. The foramen ovale presented an oval defective closure about 6 mm. in the greatest diameter. There was moderate chronic passive congestion of the liver.

CASE 6 (L. C., 152471).*—A 5-month-old female infant was brought to the Children's Hospital on Aug. 26, 1931, because of swelling of the right thigh and failure to move it for four days. The infant's mother was said to have had heart trouble since early childhood but had been able to endure two previous pregnancies without appreciable difficulty in labor. The patient had a full-term, normal delivery, and weighed 4 pounds, 11 ounces. A diagnosis of congenital heart disease was made at birth, apparently from the auscultatory cardiac signs. For two weeks before entry to the hospital, the child had been somewhat feverish and, on the day of entry, mild cyanosis about the lips was noted by the mother.

The physical examination revealed an apathetic, acutely ill infant with cyanosis of the face and extremities. The neck veins were not distended. Numerous scattered rhonchi and fine, crepitant râles were heard over both lungs. The heart was thought to be slightly enlarged, and there was a loud, precordial systolic murmur with a palpable thrill. No diastolic murmur was noted. The abdomen was normal, and the right thigh was swollen and held in the flexed position. No clubbing of the extremities was noted. A clinical diagnosis of acute osteomyelitis was made, but the patient was too ill to be subjected to surgery at the time. The temperature ranged from 103.4° to 104.6° F., and the pulse ranged from 120 to 180 per minute.

Laboratory studies revealed a hemoglobin of 65 per cent, a red blood cell count of 4.85 million, and a white blood cell count of 24,400 per cubic millimeter. The urine was normal. Fluid parenterally and supportive measures were of no avail, and the patient died, two days after entry. The clinical diagnoses were: (1) congenital heart disease, (2) osteomyelitis of the right femur, and (3) bronchopneumonia.

Autopsy Findings.—Anatomic diagnoses: (1) septicemia (*Streptococcus hemolyticus*); (2) congenital heart disease with stenosis of the pulmonary valve; (3) acute bacterial endocarditis (pulmonary valve); (4) edema, generalized; (5) acute glomerulonephritis; (6) abscesses of the lungs, myocardium, liver, and kidneys; and (7) osteomyelitis, periostitis, and epiphysitis of the right femur.

*Reported by Farber and Hubbard, 1933.[3]

The heart was prominent in the region of the right ventricle, and the weight was 30 grams, with an estimated normal weight of 29 grams. The wall of the right ventricle measured 7 mm. in thickness, as compared to that of the left ventricular wall which measured 8 millimeters. The right auricle and right ventricle were slightly dilated. The foramen ovale was closed, and the interventricular septum was intact. The pulmonary ring was markedly stenotic and measured 16 mm. in circumference. The pulmonary cusps were somewhat thickened and the surfaces roughened. The pulmonary artery was of normal size. On the anterior leaflet of the pulmonary valve, attached to the surface next to the pulmonary artery, there was a small, irregular, fresh fibrinous vegetation, 1 by 1.5 by 2 mm., which was firmly attached to the valve surface. No other vegetations were seen and the tricuspid mitral, and aortic valves were normal.

Microscopic examination of the pulmonary valve revealed thickening of the valve, with some increase in connective tissue. In the vegetation attached to the valve, numerous cocci were demonstrable. No scarring was seen in any of the other valves, or in the myocardium.

CASE 7 (H. C., 11901).—A 5-month-old male infant was brought to the Children's Hospital on May 23, 1925, because of constant crying and cyanosis of the skin. For ten days following birth, the patient was said to have been a blue baby. He was noted to be cyanotic following this, but the cyanosis was not very prominent except during crying.

The physical examination revealed a poorly-developed and poorly-nourished male infant, appearing mentally alert. The skin revealed moderate cyanosis, but no clubbing of the extremities was noted. The mucous membranes of the mouth became very cyanotic during crying. The lungs were normal by percussion and auscultation. The heart was thought to be enlarged to the right, and a loud precordial systolic murmur, not rough, was noted; this murmur was transmitted to the left axilla and the left side of the chest posteriorly. The second pulmonic sound was decreased, and a thrill was palpable at the base of the heart in the pulmonic area. The heart rhythm was regular, and the rate was 148 per minute. The abdomen was normal. The extremities were normal, except for the cyanosis. A teleroentgenogram of the chest revealed an enlarged heart shadow, measuring transversely 6.5 cm., but there was no characteristic configuration or abnormality in shape.

The laboratory studies revealed a hemoglobin of 115 per cent, a red blood cell count of 8.75 million, and a white blood cell count of 19,800 per cubic millimeter with 64 per cent lymphocytes, 34 per cent polymorphonuclears, and 2 per cent monocytes. Urinalysis was negative.

There was no change in the course of the disease until June 13, when he developed fever. A blood culture two days later contained a pure culture of streptococci. Signs of sepsis progressed, and he died June 19, 1925.

Autopsy Findings.—Anatomic diagnoses: (1) stenosis of pulmonary valve of the heart, (2) coronary sclerosis, (3) cardiac hypertrophy, (4) chronic passive congestion of the viscera, and (5) bronchopneumonia.

The heart was moderately enlarged and prominent in the region of the right ventricle. The right ventricular wall measured 1 cm. in thickness. The interventricular septum was intact and the foramen ovale was closed. The pulmonary ring was quite markedly stenosed and measured 4 mm. in circumference. There was moderate sclerosis of the coronary arteries. The pulmonary valve was not examined microscopically. The liver showed moderate chronic passive congestion, but there was no evidence of scarring.

CASE 8 (D. L., 134420).—A 15-year-old boy was admitted to the Children's Hospital in April, 1936, because of weight loss of two weeks' duration. In the preceding two years, there had been intermittent edema of the genitalia and extremities and marked mottling of the skin on exposure to the cold.

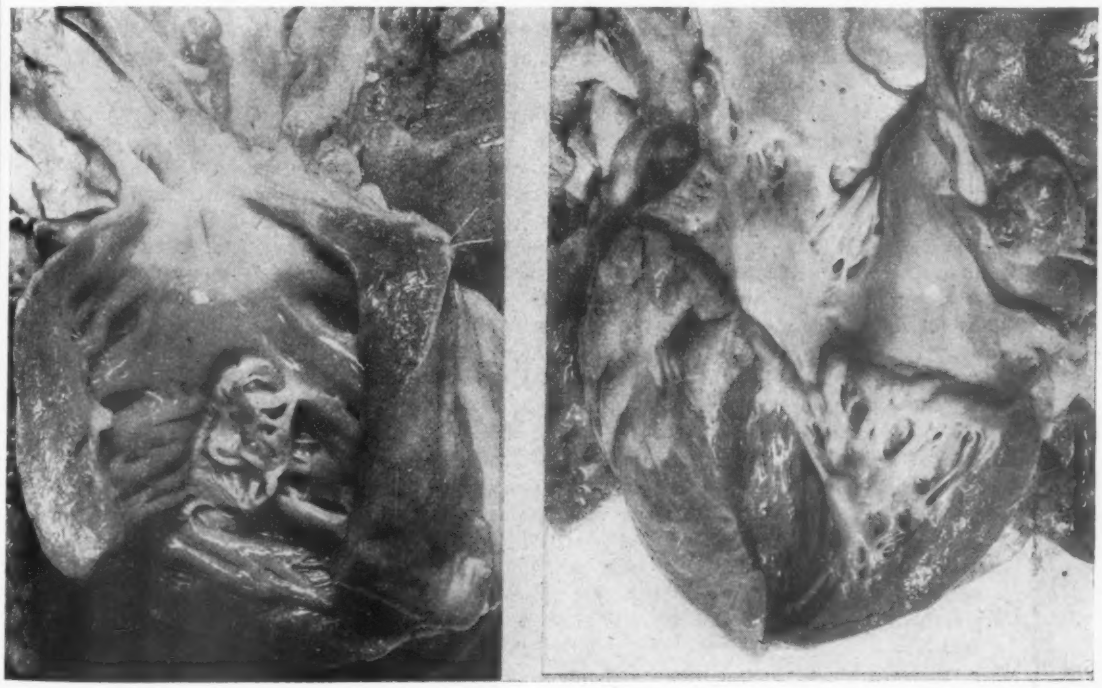
The physical examination revealed an emaciated boy with a barrel-shaped chest and a marked depression of the sternum. There was moderate scoliosis of the dorsal spine. Evidence of fluid was found in the left side of the chest. The heart sounds were of good quality, and both systolic and diastolic murmurs were described over the precordium. The spleen was enlarged, and pitting edema of the lower extremities was quite marked.

The laboratory studies revealed a normal urine, except for a Grade 1 albuminuria. The hemoglobin was 128 per cent (Sahli); the red blood cell count was 6,230,000 and the white blood cell count was 9,050 per cubic millimeter, with a normal differential count. There was no fever. The x-ray films of the chest revealed evidence of a considerable amount of fluid in the left lower lateral chest. The heart was markedly enlarged and displaced to the right.

A rather "flocculent" congestion or edema of the lungs was described. Thirty cubic centimeters of bright, sanguineous fluid was removed from the left side of the chest by thoracentesis. It was thought at the time that the pleural fluid on the left was due to congestive heart failure.

On March 8, 1937, he was readmitted to the hospital. At this time there was observed little physical development over that of his previous entry. Six months before re-entry, he had had his appendix removed under local anesthesia at another hospital. His complaints on the later entry were dyspnea and edema of the legs. A large amount of fluid was removed from the left side of the chest, on entry, a culture from which revealed *Staphylococcus aureus.* On March 14, 1937, the heart was found to be enlarged, and both systolic and diastolic murmurs were described, indistinct at the apex, and loudest in the hollow of the deformity of the chest. A thrill was palpated over the lower end of the sternum, and the pulmonary second sound equaled the aortic second sound in intensity, although it was not accentuated. The liver was moderately enlarged. The fingers were long, but there was no clubbing.

The hemoglobin, on this entry, was 90 per cent; the red blood cell count was 4,900,000 and the white blood cell count was 22,100 per cubic millimeter.



A. *B.*

Fig. 5.—The heart in Case 8. *A*, The right ventricle is open to demonstrate the moderate stenosis and scarring of the pulmonary valve and ring. The scarred tricuspid valve with the thickened chordae tendineae can be seen. The hypertrophy of the wall of the right ventricle is also apparent. *B*, The left ventricle and auricle are open to demonstrate the abnormal mitral valve, with thickening and shortening of the chordae tendineae. The fenestration of the septum secundum is also apparent.

He was then discharged home, where he carried out simple activities but was bothered by orthopnea and an occasional slight rise in temperature. He was re-admitted to the hospital on March 30, 1937, where a rib resection was carried out two days later. Following this operation his temperature remained elevated, the pulse became fast, and he died on April 6, 1937.

Autopsy Findings.—Anatomic diagnoses: (1) bacteriemia, *Str. hemolyticus;* (2) congenital heart disease with: (a) stenosis of the pulmonary valve, (b) absence of one coronary artery, (c) fenestration of the aortic valve leaflets and septum secundum, (d) hypertrophy of the right ventricle, (e) network of Chiari, and (f) scarring of the tricuspid, aortic, and mitral valves; (3) bronchopneumonia; (4) chronic pleuritis and empyema, left, old; (5) focal necrosis of the liver with bile stasis and periportal cirrhosis; (6) chronic passive congestion, generalized; and (7) pulmonary edema.

The heart was moderately enlarged, and the right ventricle was hypertrophied. The foramen ovale was closed, and the interventricular septum was intact. The pulmonary valve was moderately stenosed and measured 2.5 cm. in circumference, as compared with 3.5 cm. for the aortic valve circumference (see Fig. 5). The valve leaflets were thickened and firm. The individual leaflets were long, with deep cryptlike sinuses behind each one. The tricuspid valve was thickened, and the chordae tendineae were thickened and measured 1.2 cm. in length. The mitral valve was similarly affected (Fig. 5, *B*), but there was no stenosis of the valve. There was thickening of the leaflets of the aortic valve, but no stenosis was produced. Several small fenestrations, measuring 1 to 3 mm., were present in the anterior and posterior leaflets of the aortic valve and in the septum secundum (Fig. 5, *B*).

Microscopic examination of sections, taken from the pulmonary valve annulus, revealed abundant, highly collagenous connective tissue. Slight vascularization of this tissue was present, and an occasional vessel was surrounded by lymphocytes. The portal veins and sinusoids of the liver were congested, and there was quite a marked increase in the periportal connective tissue. Strands of connective tissue extended from one portal area to another. Moderate infiltration of this tissue with lymphocytes, monocytes, and a few polymorphonuclear leucocytes was present.

CASE 9 (B. C. H., 1014034).*—A 20-year-old white girl was first admitted to the Boston City Hospital in March, 1937, with a complaint of dyspnea on exertion of one year's duration. She had not been a "blue baby" and had no history of rheumatic fever or joint pains.

The patient was well nourished and moderately well developed. There was visible pulsation of the cervical veins. The heart was enlarged to the right and left, and the point of maximal impulse was in the fifth intercostal space, 10 cm. from the midsternal line. The right border of cardiac dullness was 4 cm. from the midsternal line in the fourth intercostal space. There was a precordial bulge, with a palpable systolic thrill felt along the left border of the sternum. There was a harsh, high-pitched systolic murmur with the point of maximum intensity at the second intercostal space, to the right of the sternum. It was transmitted widely, over the precordium, and upward, along the left side of the neck, and, slightly, along the right side of the neck. There was also a systolic "shock," palpable over the precordium. A loud systolic murmur was heard over the spine and upper chest posteriorly. The blood pressure measured 114/82, and the lungs were clear to examination. The abdomen was normal. There was moderate pretibial and ankle edema.

During her hospital stay, the laboratory data were as follows: hemoglobin, 82 per cent; red blood cell count, 3.75 million, and white blood cell count, 9,600 per cubic millimeter; sedimentation rate, 5 mm. per hour; blood Hinton test, negative; and urine normal, with a specific gravity of 1.020. No growth was obtained from two blood cultures. A teleroentgenogram of the chest was intrepreted as showing cardiac enlargement with a marked "rheumatic" deformity. The pulsations were poor during fluoroscopy. Electrocardiogram showed a normal sinus rhythm and a high degree of right axis deviation. The S-T segments were depressed in Leads II and III, and the T waves in the same leads were inverted (see Fig. 1, *D* and *E*).

She was placed on bed rest, and fluids were restricted while in the hospital; the edema subsided rapidly. She was discharged after two weeks, considerably improved.

The patient next returned to the hospital in December, 1938, eighteen months later. Six weeks before this admission, she had first noticed swelling of the abdomen followed by edema of the ankles. In addition, she had had gradually increasing dyspnea on exertion.

When examined she appeared well nourished with slight orthopnea, but without cyanosis. The neck veins were distended. Over the left lower chest posteriorly, there was dullness to percussion, diminished voice and breath sounds, and a few, medium, coarse moist râles. The heart findings were essentially the same as on the previous entry. Shifting dullness and a fluid wave were demonstrated in the abdomen. The liver and spleen were not felt. There was moderate pitting edema over the sacrum and ankles.

The venous pressure in the arms was 30 cm. of water and that in the legs, 31 cm. of water. The electrocardiogram had not changed appreciably. The x-ray heart shadow was described as globular in shape and some increased density was noted in the left lower chest, which was thought to be due to fluid. By fluoroscopic examination, marked symmetrical enlargement of the heart was noted. The cardiac pulsation in the region of the left ventricle was diminished to absent, while the pulsation in the region of the right ventricle was described as slight.

*Case 1 of Wexler and Ellis, 1944.[4]

The patient was treated with digitalis, Salyrgan, restriction of fluids, and rest in bed. The edema and ascites gradually disappeared, and she became less dyspneic, but the neck veins remained distended. The cardiac shadow did not change in size or contour during this time. She was discharged on the twenty-fourth day of her hospital stay with instructions to continue taking digitalis and gradually to resume moderate activity.

The patient again entered the hospital in October, 1940, because of increasing swelling of her abdomen and dyspnea. She had been taking 0.1 Gm. of digitalis daily, since her previous discharge and had restricted her physical activity. If the patient remained in bed, the size of her abdomen decreased; if she attempted to resume any activity, the ascites would sometimes return. The patient had not noted cyanosis, and her ankles had not been swollen.

The blood pressure was 110/72; the pulse rate was 92 per minute; the respiratory rate was 24; and the temperature was 98.6° F. The heart signs and sounds were essentially the same as during the previous admission. There were ascites and sacral edema but no ankle edema.

The laboratory studies revealed a hemoglobin of 85 per cent, a red blood cell count of 4.15 million, and a white blood cell count of 6,700 per cubic millimeter. The total protein varied between 5.3 and 5.7 Gm. per cent. The urine contained albumin, 1 to 3 plus, and many white blood cells in clumps. The venous pressure in the arms was 31 cm. of water on admission, and it gradually dropped to 19 cm., on discharge. The circulation time, by the sodium cyanide method, was 50 seconds, on admission, and 30 seconds, on discharge. Electrocardiograms and teleroentgenograms of the chest showed no significant changes from those of the previous entry. The basal metabolic rate was -2. While in the hospital she received 0.1 Gm. of digitalis every day, mercupurin, 2 c.c. every other day, and rest in bed. She repeatedly had reactions to mercupurin, which consisted of a feeling of faintness, transient dyspnea, and a feeling of impenidng disaster for a few minutes after the injection, which was always given slowly. On this regime she improved, although the ascites never completely subsided. She was discharged after fifty-two days.

In May, 1941, the patient had her final hospital admission because of dyspnea and abdominal swelling. Physical examination showed a thin girl with evidence of moderate loss of muscular substance, particularly over the upper part of her body. The heart findings were much the same as before, and she also had signs of fluid in both pleural cavities. Her abdomen was greatly distended with fluid.

The laboratory and clinical data were much the same as before, including the venous pressure values and circulation time. In addition, the total serum protein was 4.8 Gm. per cent: the globulin was 2.4 Gm. per cent, and the albumin was 2.4 Gm. per cent. Urobilinogen determinations in the urine varied between 1:1 and 1:8, the hippuric acid excretion was 1 Gm. in four hours, and the prothrombin time was 36 per cent of normal. The bromsulfalein test showed 90 per cent retention in five minutes, 45 per cent retention in 15 minutes, and 20 per cent retention in 30 minutes.

She was placed on bed rest and given digitalis and Mercupurin. In the middle of the second hospital month, auricular fibrillation appeared for the first time (Fig. 1, *E*) and was present thereafter. The ascites and pleural fluid persisted and at the end of the third month, a right thoracentesis was performed with recovery of clear yellow fluid. Culture of this fluid yielded no growth. One week later, 12 liters of a similar clear transudate fluid were removed slowly by abdominal paracentesis; this fluid was likewise sterile to culture. There were an estimated 6 liters left in the abdominal cavity. Fluid slowly reaccumulated in the right side of the chest and abdomen. The right side of the chest was again tapped, a few days later, and 1,200 c.c. of clear fluid were removed without incident. About two hours later, Mercupurin was given intravenously, as had been done on many previous occasions, and, directly after the injection, the patient's breathing became rapid and shallow. After a few seconds she developed acute opisthotonous, the heart stopped beating, and she expired.

Autopsy Findings.—Anatomic diagnoses: (1) pulmonary stenosis, congenital, with marked hypertrophy of the right auricle and ventricle; (2) mural thrombus, right auricle; (3) cirrhosis of the liver; (4) ascites and hydrothorax, right; (5) chronic passive congestion of viscera; (6) fibrocaseous pulmonary tuberculosis, involving the right apex, diaphragmatic pleura and tracheobronchial lymph nodes; (7) hyperplasia of red cell series of bone marrow; and (8) healed perisplenitis.

The body was that of an underdeveloped, poorly nourished white girl. There was no peripheral edema. Five liters of clear yellow fluid were present in the peritoneal cavity and 300 c.c. of similar fluid were found in the pericardial cavity. Two liters of blood-tinged fluid were present in the right pleural cavity, and the pleural surfaces were coated with a thin film of friable, gray-green purulent material. This was most marked over the surface of the

diaphragm. The heart weighed 380 grams. The edges of the cusps of the pulmonary valve were fused so that the valve opening measured but 0.5 cm. in diameter. The valve edges were slightly thickened but were smooth and showed no evidence of calcification or of inflammation (Fig. 6). The bodies, bases, and commissures of the valve were not remarkable. The pulmonic ring at the base of the valve measured 5 cm. in circumference. Microscopic sections, taken from the base of a pulmonary valve cusp, showed no abnormalities. The right ventricle was hypertrophied, so that the wall measured 1.8 cm. in thickness. The papillary muscles were somewhat flattened and the ventricle appeared dilated. The right auricle was markedly dilated and, attached to the wall of the right auricular appendage, was a soft gray-red thrombus. The wall of the left ventricle measured 1 cm. in thickness, and the chamber was not remarkable. The left auricle was not enlarged. The tricuspid, mitral, and aortic valves and the coronary vessels were negative. The right lung weighed 200 grams. There was a

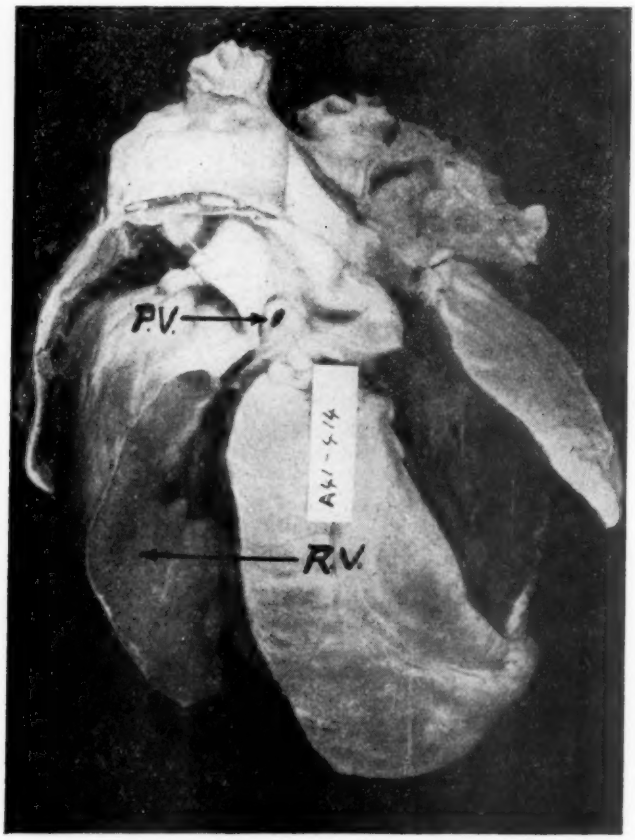


Fig. 6.—The heart in Case 9, with the right ventricle open. The marked hypertrophy of the right ventricular wall (R.V.) is apparent. The pulmonary valve leaflets were fused together forming a dome at the top of which was the small aperture (P.V.) The pulmonary annulus was not particularly stenosed.

firm, gray nodule 0.3 cm. in diameter at its apex, which, on histologic examination, showed typical fibrocaseous tuberculosis. The tracheobronchial lymph nodes were moderately enlarged and contained caseous nodules, which were also demonstrated by histologic sections to be due to tuberculosis. The left lung weighed 120 grams and was negative upon examination. The liver weighed, 1,090 grams. The surface was red-brown in color and coarsely and irregularly nodular. The parenchyma was divided into rough, irregular nodules by bands of firm, gray-white fibrous tissue. Microscopic examination showed extensive replacement of liver cells by broad bands of scar tissue. There was marked congestion about the central veins, and the liver cells around many of these veins were replaced by fibrous tissue. The spleen weighed 160 grams and was firm in consistency. The combined weight of the kidneys was 250 grams. No abnormal changes were found on gross and microscopic examinations. The bone marrow showed moderate hyperplasia of blood cells of the erythrocytic series.

CASE 10 (A. W., A09-107).—A 34-year-old housewife entered the Boston City Hospital in May, 1906, because of dyspnea, abdominal swelling, and edema of the lower extremities of three months' duration.

At the time of admission, the temperature was 98° F.; the pulse rate was 100 to 140 per minute; and the respirations were 25 to 40. The patient was well nourished and of normal development. Edema was described as generalized, and the patient suffered from dyspnea and orthopnea. There was moderate exophthalmos. The neck veins were slightly distended. The heart was percussed 4.5 cm. to the right of the sternum. The upper and left cardiac borders could not be identified. The apex beat was felt in the sixth intercostal space, slightly to the left of the midclavicular line. The heart action was irregular in force and rhythm. The first heart sound was sharp and was preceded at the apex by a presystolic "roll." There was a ringing second sound at the base, just to the left of the sternum. A presystolic thrill was felt at the apex, and a systolic thrill was present at the base and, at times, over the entire precordium. There was a loud systolic murmur heard at the base of the heart and this was most marked just to the left of the midsternum. There were a systolic murmur and a diastolic bruit at a point slightly below the middle third of the left clavicle. A to-and-fro pericardial friction rub was heard at the apex and, at times, over the midprecordium. The radial pulses were small and irregular in force and rhythm. The cardiohepatic angle was normal. There was slight dullness to percussion over the posterior portion of the right lower lung, and moist râles were heard by auscultation of the right base. Anteriorly, the entire left lung was flat by percussion, and there was distal bronchial breathing and absent tactile fremitus. The posterior portion of the left lung was dull by percussion over the upper half; the lower half was flat by percussion, and the breath sounds were diminished. An occasional moist râle was present at the left lung base. The abdomen was distended and tense. The abdominal wall was edematous. The flanks were flat, when percussed, and a fluid wave and shifting dullness were demonstrated. The liver was palpated 4 fingerbreadths below the right costal margin. There was marked edema of the legs. Several superficial excoriations and ulcerations were present on the right lower leg. There was a papillary eruption about the elbows and on the back. The knee jerks were not obtained.

Urinalysis revealed a specific gravity of 1.012, a slight amount of albumin, and numerous hyaline and granular casts.

The patient was given digitalis. On the second hospital day approximately 2,000 c.c. of fluid were removed from the left side of the chest. The patient's condition improved for a few days but then became progressively worse; she died on the twelfth hospital day.

Autopsy Findings.—Anatomic diagnoses: (1) pulmonary stenosis, congenital, with marked hypertrophy of the right auricle and ventricle; (2) mural thrombus (right auricle) ball thrombi, (left ventricle) obliterative pericarditis; (4) congestive cirrhosis of the liver; (5) ascites (bilateral hydrothorax); (6) atelectasis (left lung); (7) infarct of the right kidney; and (8) leiomyomata of the uterus.

There was marked edema of the lower extremities and moderate edema of the chest wall and face. There were numerous sharply demarcated, circumscribed ulcers over the lower extremities. The peritoneal cavity contained approximately 3 liters of clear serous fluid. One liter of similar fluid was present in the left pleural cavity, while 500 c.c. of fluid were present in the right pleural cavity. The pericardial cavity was obliterated by dense inelastic adhesions, except over the right auricle. The heart weighed 520 grams. The edges of the cusps of the pulmonary valve were interadherent for approximately one-third of their total length, with consequent narrowing of the functional opening to 1 cm. in diameter. The valve cusps were not thickened and the valve sinuses were deep. There were a few small areas on the free margin of the tricuspid valve which were thickened. The leaflets of the mitral valve were slightly thickened, along their line of closure, and along their free margins. The aortic valve was normal. The right auricle was approximately three times the usual size and was markedly dilated. In the auricular appendage was a large, firmly adherent mass of mottled red and gray clot. The right ventricle was hypertrophied; its wall measured 1.5 cm. in thickness, and the papillary muscles were larger than those of the left ventricle. The left auricle was not remarkable. The wall of the left ventricle was thickened and measured 1.75 cm. in width. Two red and white, laminated ovoid masses, with uniformly smooth surfaces, were lying free in the cavity of the left ventricle. These measured 2 by 1 by 1 and 1 by 1 by 0.5 centimeters. Throughout the ventricular muscle of both cavities were areas of pale yellow and dark red mottling and, in many places, opaque, white, irregular areas. The coronary sinus was dilated so that the forefinger could be passed readily through the first few centimeters of its length. The coronary arteries were not remarkable. Microscopic sections showed focal areas of replacement of the myocardium by scar tissue. The liver weighed 1,500 grams.

The surface was nodular, but soft, and could be cut without undue resistance. The parenchyma was mottled with large, irregular areas of dark red and bright yellow. No lobular markings could be distinguished. The hepatic veins were greatly dilated.

Microscopic sections of the liver showed a marked increase in connective tissue. This tissue was arranged, in some areas, in the form of dense circumscribed bands and, in other areas, as irregularly branching, dense masses. These masses contained isolated groups of liver cells, bile ducts, and blood vessels. The liver sinusoids were considerably dilated and many of the liver cell columns were shrunken.

CASE 11 (W. J. H.).—An 11-year-old boy entered the Boston City Hospital on April 10, 1910, complaining of headache and eye trouble of one week's duration. The headache had become progressively worse. Apparently, also for the first time, he had noted mild dyspnea on exertion and cyanosis during the one week before entry. For two days there had been moderate drowsiness. The family and past histories were irrelevant.

The physical examination revealed a semistuporous boy who was irritable when disturbed. There was paresis of the muscles of the right side of the face and ptosis of the left eyelid. The lungs were clear to examination. The examination of the heart revealed a rough systolic murmur, heard all over the precordium, but it was loudest at the base, where there was an associated thrill. There was no paralysis of the extremities. The temperature was 99° F. The pulse rate ranged from 60 to 90 per minute, and the respirations were 20 per minute. When a lumbar puncture was done, clear fluid was obtained. Two days after entry the patient suddenly became cyanotic; the respirations became gasping in character and then ceased. The heart action continued for a short time after respiratory movement had stopped. The clinical diagnoses of brain abscess and pulmonary stenosis were made.

Autopsy Findings.—Anatomic diagnoses: (1) cerebral abscess; (2) pulmonary stenosis, congenital; (3) hypertrophy of the right ventricle; (4) chronic passive congestion of the liver and kidneys; and (5) patent foramen ovale.

The heart was enlarged, weighing 340 grams; there was particular prominence of the right ventricle. The left auricle and ventricle were normal. The pulmonary valve ring measured 4 cm. in circumference while the aortic valve measured 5 centimeters. The pulmonary cusps were uniformly about 1 mm. thick and presented unusual stiffness, with slight irregularity. No calcification was found. The cusps were fused along the edges so that the outlines were nearly obliterated and a buttonhole orifice, 9 mm. long was produced. The tricuspid, mitral, and aortic valves were normal. The foramen ovale was patent and measured 1.1 by 0.3 centimeters. The wall of the right ventricle was markedly thickened and measured 2.5 cm. across, while the wall of the left ventricle was the usual size and measured 1 centimeter. The interventricular septum was intact. The lungs and liver were normal except for moderate congestion. In the left temporo-occipital lobe of the brain there was an abscess measuring 4.5 cm. in diameter, which contained foul-smelling pus. Microscopic examination of sections of the myocardium were negative.

DISCUSSION AND COMMENTS

It is apparent in reviewing the cases (Table I) that, although the degree of pulmonary stenosis varied somewhat, there was moderate to marked stenosis of the pulmonary valve, or pulmonary annulus, in all except Case 4. It is also apparent that the degree of right ventricular hypertrophy was a good index as to the degree of stenosis. There seemed to be two types of stenosis, one of which affected, primarily, the pulmonary annulus with marked scarring of the annulus and the valve leaflets. This type of stenosis predominated in nine cases. The other type was the result of fusion of the edges of the valve leaflets to form a dome, with an aperture at the peak of the dome. This was present in two cases and was best exemplified in Case 9. Similar cases have been reported.[5, 6] The right auricle was not infrequently dilated and, in three cases, mural thrombi were found therein. No patient was observed, however, to have suffered from pulmonary embolism or infarction.

The age incidence varied from infancy (three cases) to 43 years, although in the latter case there was only a mild degree of pulmonary stenosis. Two patients lived to the fourth decade with quite a high degree of pulmonary stenosis.

TABLE I

CASES	AGE (YRS.)	SEX	DYSPNEA	SYSTOLIC MURMUR	CYANOSIS	PATENT FORAMEN OVALE	DEGREE OF PULMONARY STENOSIS	RIGHT VENTRICULAR HYPERTROPHY	DILATED PULMONARY ARTERY	CAUSE OF DEATH	REMARKS
1	18	M	+++	+++	++++	++	+++	+++	+	Heart failure	
2	4.5 months	F	0	+++	0	+	+++		+	Subdural hematoma	Talipes varus
3	22	F	+++	+++	+++	++	+++	+++	0	Heart failure	Right auricular and ventricular thrombi, large right auricle
4	43	F	0	++	+	0	+	+	0	Carcinoma, pellagra	
5	30	M	++	+++	++	+	+++	+++	0	Pneumonia	
6	5 months	F	0	+++	+	0	+++	++	0	Osteomyelitis, septicemia	
7	5 months	M	0	+++	++	0	+++	+++	0	Septicemia	
8	16	M	++	++	+	0	+++	+++	0	Heart failure, septicemia	Depressed sternum, hepatic cirrhosis
9	24	F	+++	+++	0	0	++++	++++	0	Heart failure, mercurial reaction	Hepatic cirrhosis
10	34	F	+++	+++	0	0	+++	+++	0	Heart failure	Right auricular thrombi, hepatic cirrhosis
11	11	M	+	+++	+	++	++	++	0	Cerebral abscess	

There was no definite preponderance of either sex, the females numbered six and the males, five.

A systolic murmur was present in each case and there was a high correlation between the intensity of the murmur and the degree of stenosis. The intensity of the murmur was usually described as maximal at the base of the heart, along the left sternal border in the second and third intercostal spaces. It was also well heard, generally, over the precordium. The intensity of the murmur, together with the lack of change of the character of the murmur with change in position and respiration, enabled one to differentiate it from the common physiologic pulmonary systolic murmur. The transmission of the murmur was likewise important. The murmur was well heard in the cervical region, particularly on the left side, and was well transmitted to the posterior portion of the chest and usually maximally in the left lower scapular region. This transmission of the murmur would tend to differentiate it from the murmur of a patient with an interventricular septal defect. In the latter case, the murmur is transmitted much less well upwards, or to the posterior chest wall. An interauricular septal defect was considered in the differential diagnosis of the murmur in a few cases but the intensity of the murmur was considerably greater in the cases of pulmonary stenosis than one would expect in cases with an interauricular septal defect. A thrill was frequently noted in the pulmonic area and was more frequent in the cases with the more intense murmurs. In two cases (Cases 4 and 8) diastolic murmurs were described in the pulmonic area, which indicated some regurgitation through the deformed pulmonary valve. The apical diastolic murmur described in Case 10 is difficult to reconcile with the anatomic findings. It is possible that ventricular dilatation, as in acute or subacute rheumatic myocardial involvement without mitral stenosis, might have caused it. The second heart sound in the pulmonic area was heard in all the cases, but was described as being diminished in intensity in four cases. It is difficult to appreciate how the second heart sound could have had its origin in the pulmonary valve in some cases of marked scarring of the annulus. This raises the question as to whether the second sound heard in the pulmonic area may not have, in part at least, been transmitted from the aortic valve. This, in fact, is quite probable. In two cases the second pulmonic sound was described as accentuated.

Cyanosis was of a significant degree in four cases, while in the remaining cases it was negligible. Cyanosis was more common, and of a higher degree, in the patients with patency of the foramen ovale. Of the 11 patients, five had a variable degree of patency of the foramen ovale. In Case 1, the patient probably had the most pronounced cyanosis, which was not of a high degree, however, until the last six years of his life. It was during this time that the pressure on the right side of the heart doubtless increased and resulted in a flow of blood from the right auricle to the left auricle, mixing venous blood with arterial blood. The relatively late onset of cyanosis, which progresses, has been noted previously in cases with pulmonary stenosis.[7]

Congestive heart failure was the chief or contributing cause of death in only five of the cases presented here. The three infants all died of causes other than heart failure, although one patient showed evidence of early acute bacterial endocarditis (Case 6). This suggests that pulmonary stenosis is usually compatible with life until the third or fourth decade, when congestive heart failure becomes severe. The patients with the congestive heart failure were also the patients with significant dyspnea. Pleural effusion was found in only two cases. The circulation time was measured in one case and was considerably prolonged,

which indicated that the circulation from the arm into the pulmonary artery was slow, since there was little pulmonary congestion to slow the circulation through the lung.

One patient had minimal pulmonary tuberculosis. We were thus unable to confirm, in this series, the high incidence of pulmonary tuberculosis that has been reported to be associated with pulmonary stenosis.[8]

The electrocardiograms which were taken in four cases showed a high degree of right axis deviation in the three cases in which there was marked hypertrophy of the right ventricle; in the fourth patient, with less pulmonic stenosis, the situation was complicated by avitaminosis. The degree of right axis deviation was similar to that seen in cases with the tetralogy of Fallot and served to differentiate these cases from those with an isolated patency of the interventricular septum in which the electrical axis is generally normal. In the fourth case the electrical axis was normal and there was only slight hypertrophy of the right ventricle. The intraventricular conduction was normal, although right bundle branch block has been noted in pulmonary stenosis.[6]

There was no characteristic configuration of the heart by x-ray examination in the several patients who were studied by fluoroscopy and roentgenography. The heart was generally enlarged in all the patients studied, and in Case 1 there was significant dilatation of the pulmonary artery. This was, perhaps, a result of the presence of a patent foramen ovale. Dilatation of the pulmonary artery was noted in one other case in which there was likewise a patent foramen ovale. In at least two of the sixteen patients studied by Abbott in which pulmonary stenosis was complicated by patency of the foramen ovale, dilatation of the pulmonary artery was noted. Dilatation of the pulmonary artery is common, of course, in patients with isolated interauricular septal defects but in such cases, in contrast to these herewith presented, there is a clear cause, namely, the overloading of the pulmonary circulation. Pulmonary regurgitation, obviously present in a few of these cases of pulmonary stenosis, and quite possibly not demonstrable clinically in others, may be a factor.

The controversial concept of fetal endocarditis has been recently reviewed by Gross.[9] The accumulating evidence from case reports,[10] and from critical analysis, casts considerable doubt as to whether such a disease exists. To our knowledge, no observations of the acute inflammatory disease have been reported, and the etiological agent is hypothetical. A more likely concept is that of a developmental defect, since convincing evidence of inflammation is not present. This may be, as Gross suggests, a result of arrest or closure of arteries, resulting in bland infarcts and fibrosis. In the several heart valves of this series which were examined microscopically there was moderate fibrosis, but no other convincing stigmas of a previous infectious disease. Another point in favor of anomalous or defective development is the presence of other developmental defects in the heart or elsewhere in the body. In two cases of the present series there was another congenital anomaly present (Cases 2 and 8). Five patients (Table I) had patency of the foramen ovale, and one patient (Case 8) had fenestration of the septum secundum, absence of one coronary artery, and scarring of the other three valves of the heart. The fusion of the cusp edges, with the formation of a dome with a central aperture, in Case 9, was unusual and is similar to other cases reported.[5, 6]

The recent observation[11] of the association of congenital cataracts and congenital heart disease in children whose mothers contracted rubella in the first two months of pregnancy is of interest in relation to congenital heart disease. The reports do not specify the type of congenital heart disease present, and this

question will have to await further elucidation. The observations give considerable support to the view that some types of congenital heart disease may result from an exanthematous disease occurring in the mother and affecting the fetus during the early months of pregnancy. The histories of the mothers of the patients presented in this series of cases are lacking in specific evidence for such an infection, although such may have been present in some. The importance of questioning the mothers of patients with congenital heart disease, regarding the occurrence of exanthemata during pregnancy, is evident.

Sufficient scarring of the liver was present in three cases to warrant a diagnosis of hepatic cirrhosis. In Case 9 the scarring was marked. Since only five of the patients in this series had heart failure, the presence of three cases of congestive hepatic cirrhosis is significant. Pulmonary stenosis with chronic heart failure, then, may be considered, along with chronic constrictive pericarditis, to be associated with a relatively high incidence of congestive cirrhosis of the liver.[12]

SUMMARY

1. Eleven cases are reported herein of stenosis of the pulmonary valve in which the interventricular septum has been intact. In five of the patients there was associated patency of the foramen ovale of variable size. In ten of the cases the pulmonary stenosis was of sufficient degree to result in moderate to marked hypertrophy of the right ventricle.

2. Electrocardiographic and roentgenographic data are presented and discussed. A high degree of right axis deviation in the electrocardiogram is characteristic.

3. Heart failure was the cause of death in five of the patients, while causes unrelated to the heart accounted for the other deaths, including that of the three infants. Hepatic cirrhosis was observed in three of the five patients dying of heart failure.

4. The etiology of pulmonary stenosis and congenital heart disease is discussed.

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A NOTE ON THE SEROLOGIC CLASSIFICATION OF STREPTOCOCCI ISOLATED FROM SUBACUTE BACTERIAL ENDOCARDITIS

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SINCE its introduction by Thiercelin[1] the term "enterococcus" has been variously applied as a synonym for *Streptococcus fecalis* and as a loose term including all fecal streptococci which exhibit one or more of the characteristics usually attributed to this group of microorganisms. Andrews and Horder[2] pointed out that such streptococci are isolated occasionally from cases of subacute bacterial endocarditis and other human infections. More recently, with respect to the strepotcocci isolated from subacute endocarditis, the classifications most frequently encountered in the literature are "alpha" or simply "green-producing" streptococci. Bacterial endocarditis due to the "enterococci" has been recorded in a few instances; Skinner and Edwards,[3] in a recent survey of the literature, cited thirty-seven cases, and Moran[4] reported twenty cases in which the streptococci isolated were probably "enterococci."

The bacteriologic methods employed in the identification of "alpha" or "green-producing" streptococci usually indicate nothing as to the exact identity of the strain. The classification, "enterococci," as pointed out by Sherman,[5] also is based on biologic features which are characteristic of, but not limited to, the group of organisms in question. Lancefield[6] and Sherman[5] reported that those organisms previously classified as "enterococci," despite marked biochemical differences, contain a common group antigen which places them in Group D (Lancefield). It has been observed over a period of years in this laboratory that strains which would have been classified as *Str. viridans* by methods other than group precipitation tests frequently belong to Lancefield Group D.

Since many of the cases of endocarditis reported as due to "alpha" streptococci or "enterococci" were studied before the development of group precipitation techniques, there are few data available on the serologic classification of streptococci isolated from the blood cultures and other sources on cases of subacute bacterial endocarditis. Skinner and Edwards[3] cited one case in which the streptococci isolated were shown to belong to Group D and added two more from their own observation. More recently, Rantz and Kirby[7] described three cases as being due to Group D streptococci, and Wheeler and Foley[8] added four more cases.

During the past two years, the streptococci isolated from seventeen additional cases, diagnosed as subacute bacterial endocarditis in various local hospitals, have been studied in this laboratory. In addition to the serologic classification of the strains isolated from these cases, the reaction on blood agar was

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determined on Huntoon's unenriched hormone agar,[9] containing 5 per cent sterile, defibrinated horse blood incubated aerobically, anaerobically, and under partial carbon dioxide tension (10 per cent) at 37° C. for forty-eight hours. In general, no consistent qualitative differences in blood agar reaction on media incubated in the various atmospheres were noted. Since the blood agar reaction depends on so many factors other than the biology of the cell and is relatively inconsistent and unstable for many non-Group A streptococci,[10, 11] each strain was tested for the production of soluble hemolysin.[12] A volume of 0.5 ml. of a ten to sixteen-hour culture in streptococcus toxin broth[13] was combined with 0.5 ml. of a 5 per cent suspension of defibrinated, washed rabbit erythrocytes and incubated in a water bath at 37° C. for two hours, centrifuged, and examined for evidence of hemolysis. Rabbit erythrocytes and saline and controls with known hemolytic and nonhemolytic streptococci (Lancefield strains J/17/A-4 and 0/90-R, respectively) were included. None of the test strains produced soluble hemolysin.

All strains were tested for heat resistance.[5] Samples of 1 ml. of ten to sixteen-hour broth cultures were incubated in a water bath at 62° to 63° C. for one-half hour, allowed to cool, then streaked on 5 per cent horse blood agar and incubated aerobically at 37° C. for forty-eight hours.

The results of these tests are summarized in Table I, together with the serologic classification of these strains, as well as the four previously reported by Wheeler and Foley.[8] Four distinct serologic types have been recognized within the Lancefield Group D in this laboratory.[11] Since there seemed to be no correlation between serologic type and biochemical activity, various other biochemical tests which were done have been omitted from Table I.

As pointed out by Lancefield,[10] hemolytic activity is not as closely correlated with the serologic group as was first supposed. The existence of nonbeta strains, serologically identical with beta strains within a given serologic group, is well illustrated in the small series recorded in Table I. The ability to withstand high temperatures, long considered characteristic of the "enterococci,"[14] is not a sufficient differential criterion, as indicated in Table I, when the "enterococci" are considered as a serologic group.

From these data it would appear that strains which are assigned such classifications as "alpha" or "green-producing" streptococci, when isolated

TABLE I. SEROLOGIC CLASSIFICATION OF STREPTOCOCCI ISOLATED FROM TWENTY-ONE CASES DIAGNOSED AS SUBACUTE BACTERIAL ENDOCARDITIS

AGE OF PATIENTS	SOURCE OF CULTURE	NUMBER OF CASES	REACTION ON 5% HORSE BLOOD AGAR	HEAT RESISTANCE	SEROLOGIC GROUP	CLASSIFICATION TYPE
Adults	Post mortem, blood culture	1			D	H69D–5
	spleen	1	Alpha	–	D	H69D–5
	mitral valve	1			D	H69D–5
	blood culture	4			D	Lanc–3
	Ante mortem, blood culture	2	Alpha	–	D	H69D–5
	blood culture	1	Beta	+	D	Lanc–1
	blood culture	2	Alpha	+	D	Lanc–3
	blood culture	1	Alpha	–	D	Lanc–3
	blood culture	1	Alpha	+	D	D–76
	blood culture	2	Alpha	–	–*	–
	joint fluid	1	Alpha	–	–*	–
Children	Ante mortem, blood culture	2	Alpha	–	D	H69D–5
	blood culture	1	Gamma	–	D	H69D–5
	blood culture	1	Alpha	–	–*	–

All strains insoluble in bile, none produced soluble hemolysin.

All strains except * reduced methylene blue.

*Classified tentatively as *Str. viridans* on basis of failure of hydrochloric acid extract (concentrated) to react with antisera for Lancefield Groups A to M.

from subacute bacterial endocarditis, are often serologically identical and frequently, as do most of the "enterococci," belong to Lancefield Group D.

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Clinical Reports

AORTIC HYPOPLASIA WITH ASSOCIATED VASCULAR AND GENITOURINARY ANOMALIES

Report of a Case With Autopsy

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HYPOPLASIA of the aortic system has been defined by Maude Abbott[1] as "that condition in which the lumen of the arterial vessels in the greater circulation remains abnormally small and the walls unnaturally thin and elastic." First described by Morgagni, in 1761, it received the attention of such men as Virchow, and is reported by Ikeda[3] to have been the subject of some one hundred case reports by 1907. Since that time only sporadic cases have been reported,[4, 5] and, since 1933, the only report of which the authors are aware is that of Werley, Waite, and Kelsey.[6] No attempt will be made to review the literature, which has been adequately reported in the papers of Burke,[2] Ikeda,[3] and Werley.[6] It is our purpose, however, to report an unusual case in which the presenting complaints were neurological, and in which a severe degree of cardiac insufficiency in a young man was apparently the result of a hypoplastic and anomalous arterial system.

CASE REPORT

History.—W. S. (Case 216553), a 31-year-old, white man, was admitted to the Strong Memorial Hospital on March 27, 1944, with a chief complaint of paralysis of the right side of the body of three weeks' duration. The patient stated that he had not felt well since the summer of 1942. At that time he was in Florida, and noted the gradual onset of fatigability, weight loss, lassitude, palpitation, and some numbness and tingling of the arms and legs. These symptoms continued, and in the spring of 1943 he returned to his truck farm outside Ithaca, New York. No history suggestive of any tropical disease could be elicited. Lassitude, anorexia, and decreased tolerance for work persisted, and, on Feb. 1, 1944, the patient consulted Dr. Simon Schmal of Ithaca. Examination at that time, according to Dr. Schmal's report, showed a pulse rate of 104 per minute and a blood pressure measurement of 105/80. The hemoglobin was 100 per cent, and serologic examination and urinalysis were negative. The patient returned on February 15, his systemic symptoms slightly improved, but he complained of deafness in the right ear, mumbled speech, and paresthesias of the right hand and arm. These had developed following a severe emotional upset. In the two days following this visit, several "nervous spells" occurred, during which, for a short time, he was unable to talk.

On March 5, 1944, about three weeks later, the patient developed aphasia, difficulty in swallowing, and a complete right hemiplegia, without loss of consciousness. He was referred to Strong Memorial Hospital for study on March 15, but on the day before his scheduled admission to the hospital, he had an episode of unconsciousness and severe coughing with production of one-half cupful of dark red blood. His temperature rose to 104° F., and he was acutely ill. Roentgenograms showed infiltrations in both lobes of the left lung and marked cardiac enlargement. A diagnosis of bronchopneumonia was made at the time, and a second x-ray examination, one week later, revealed definite clearing of the infiltrations and a reduc-

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tion in heart size. During the acute stage of the illness, signs of heart failure developed, and he was given small doses of digitalis. By March 27, the patient was deemed sufficiently recovered for transfer to Rochester.

The past history contained few positive findings. There had been a "tuberculous gland in the neck" when the patient was 18 months old and what was apparently an uncomplicated pneumonia ten years before admission. Otherwise, he had led an active, vigorous, outdoor life, and had done considerable mountain climbing as a pastime. At various times he had been a preacher, farmer, and somewhat of a recluse. There was no history of rheumatic fever or chorea, and there had been no dyspnea, orthopnea, or ankle edema prior to the present illness. It was noted on admission that the patient's voice was unusually high-pitched, and members of the family declared it had always been so.

There was no family history of nervous disorders or congenital anomalies of any sort. The father and mother were living and in reasonably good health, though the patient's mother had hypertension. A sister was living and well.

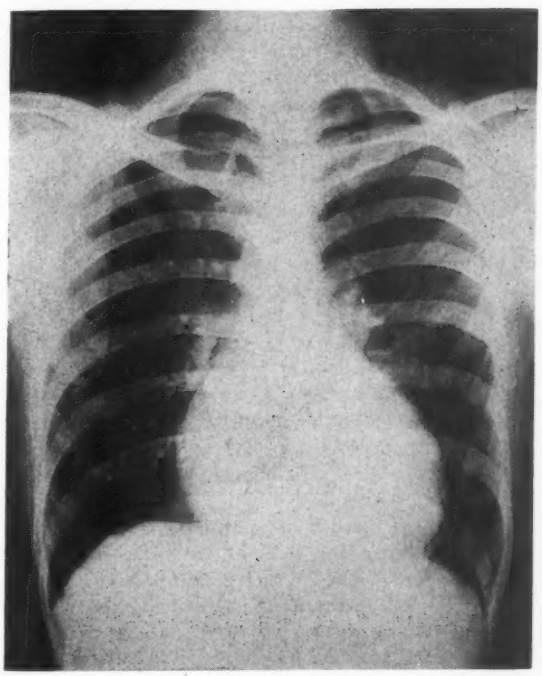

Fig. 1.—Teleroentgenogram taken March 28, 1944. Note the enlarged globular heart with small inconspicuous aorta.

Physical Examination.—On admission the temperature was 37° C., the pulse was 82 per minute, the respirations were 16 per minute, and the blood pressure measured 130/88 in both arms. The patient was a malnourished individual who appeared chronically ill and could not move the right arm or leg. The voice was unusually high pitched. The patient was oriented and cooperative. The skin was dry and coarse. No petechiae were noted, and there was normal distribution of body hair. The left pupil was somewhat smaller than the right, but both reacted to light and accommodation. The fundi were normal. The tongue was somewhat redder than normal, and cheilosis was noted at the corners of the mouth. There were no lesions on the buccal mucous membranes. The trachea was in the midline. The lungs were clear to percussion and auscultation. The cardiac point of maximal impulse was in the left anterior axillary line, and the heart was diffusely enlarged in nondescript fashion. The heart was overactive, and the sounds were loud. No thrills, rubs, murmers, or irregularities were detected. No abdominal organs were palpable, and there was no tenderness. No sacral edema was noted. Both testicles were in the scrotum, and both were thought to be of normal size and consistency. The left foot was somewhat colder than the right. The dorsalis pedis pulsation was absent on the left, while the posterior tibial and popliteal pulsations were only barely palpable. All peripheral pulsations were of normal strength in the right leg. The blood pressure was 110/70 in the left leg and could not be obtained in the right. The left

radial pulse was appreciably weaker than the right and the radial artery was in a somewhat anomalous position. There was considerable atrophy of the interosseous muscles of the right hand and the muscles of the right arm. The neurological examination revealed a right hemiplegia. A right lower facial paralysis was apparent. The tongue deviated to the right. Abdominal and cremasteric reflexes were present on the left, but none could be elicited on the right. The deep reflexes were hyperactive on the right side, while the ankle jerk and knee jerk were absent on the left. There was a positive Hoffman's sign on the right but Babinski's sign was absent. Some motor aphasia was present.

Laboratory Findings and Course in the Hospital.—The Wassermann test was negative. The erythrocyte count was 4 million and the hemoglobin 13 Gm. The leucocyte count was 9,000 per cubic millimeter, with 70 per cent polymorphonuclear cells and no eosinophiles. The urinary findings were negative except for a rare red blood cell in the sediment, and the findings continued similar throughout the patient's stay in the hospital. The examination of the stool gave a negative test for blood, and no parasites were found, either on direct examination or by the zinc flotation method. A lumbar puncture was done, and the spinal fluid showed no increase in cells or abnormalities in dynamics. The spinal fluid serologic test was negative, and the total protein was 25 mg. per cent. Numerous roentgenograms were taken, and the following findings were reported: *Posteroanterior film of the chest:* The heart was enlarged, globular in shape, with well-rounded borders. The transverse diameter was 15.8 centimeters. The transverse diameter of the chest, at the level of the diaphragm, was 26.8 centimeters. The aorta was inconspicuous and not visualized beyond the shadow of the spine or sternum. *Fluoroscopy:* Fluoroscopy in the supine position indicated all chambers share in the cardiac enlargement. Good pulsations were present at all borders. The aorta was not visualized, and no aortic indentation was seen on the barium-filled esophagus. *Skull:* Negative examination. *Abdomen* (film of kidneys, ureter, and bladder): The left kidney was outlined normally. The right psoas muscle and the right kidney were not visualized.

Two electrocardiograms were taken and showed extreme right axis deviation, the first tracing having an axis of 150 degrees and the second 80 degrees. Aside from a tachycardia of 120 per minute in the second tracing, there were no other significant findings. Repeated blood cultures were negative, and a muscle biopsy, taken from one gastrocnemius muscle, showed no evidence of periarteritis nodosa. Circulation times were measured but were considered inaccurate because of undependable responses from the patient.

The patient had an essentially afebrile and uneventful course for the first twelve days of admission, and injection of diodrast was contemplated for the purpose of visualizing the aorta. The only change in this period was recovery of a minimal amount of function in the right leg. On April 10, however, the patient began to feel poorly and coughed up a small amount of fresh blood. Some cyanosis of lips and nailbeds was noted. The temperature and pulse increased, and a loud gallop rhythm was heard over the sternum. A roentgenogram taken at this time showed an infiltration in the right lung field, thought probably to be an infarct. A sputum examination revealed only *Streptococcus viridans, Staphylococcus albus,* and diphtheroids bacilli. The temperature continued to rise, and the administration of sulfadiazine was commenced. The pulse was maintained at about 120 per minute. Icterus developed, and it was felt that the patient was having multiple pulmonary infarcts. Medication with sulfadiazine was discontinued, and, during the last twenty-four hours, signs of cardiac failure were noted. The patient was digitalized and placed in an oxygen tent, but he expired on April 17, 1944.

Autopsy Findings (Case A8542).—A complete autopsy was done. All organs were carefully studied, but only the gross pathologic changes and pertinent negative findings are presented.

The body was that of an extremely emaciated 31-year-old white man. The skin was jaundiced, particularly over the face and neck, and the sclerae showed pronounced icterus. The left pupil was 2 mm. in diameter, and the right, 4 millimeters. There was no clubbing of the fingers and no edema. The peritoneal cavity contained about 150 c.c. of clear, light yellow fluid. The left pleural cavity contained about 600 c.c. of cloudy, yellowish-brown fluid, and the right pleural cavity about 100 c.c. of the same type of fluid. There were a few easily broken adhesions, bilaterally. The pericardial cavity contained about 125 c.c. of light yellow, slightly cloudy fluid.

The heart weighed 490 grams. The subepicardial fat was somewhat decreased in amount. There were two or three small soldier's plaques which were less than 1 mm. thick and did not extend into the musculature. The chambers were all extremely dilated, and the ventricles were saucer-shaped. In the auricular appendages of the right heart and adherent to the muscular trabeculations of the left ventricle, there were a few small thrombi. In the

center of the foramen ovale, there was a small perforation about 1 mm. in diameter. The ductus arteriosus was demonstrated to be closed. The endocardium was smooth throughout. Beneath the surface of the ventricular endocardium there were a few, diffusely scattered, yellowish, sharply circumscribed areas, 1 to 3 mm. in diameter. These were less than 1 mm. thick but extended somewhat into the musculature. The tricuspid and mitral valves were dilated, but all valves were normal. The left ventricular wall was 0.8 cm. in thickness, and the right was 0.3 centimeter. There were no areas of infarction or thrombosis. The coronary arteries showed no abnormalities.

The aorta was of good elasticity and showed no degenerative changes. The aortic valve was 7.5 cm. in circumference. The aorta, immediately above the aortic valve, was 5 cm. in circumference. At the apex of the arch, it measured 3.2 cm., and in the distal descending portion of the arch, it measured 3.2 centimeters. The caliber became narrower where branches in the thoracic and abdominal cavities were given off. At about the level of the third lumbar vertebra, it bifurcated. The left branch was about 1.2 cm. in circumference, and the right branch about 0.6 centimeter. The right branch appeared to represent the right internal spermatic artery. Its main portion continued in the normal course of the spermatic artery but, about 10 cm. from its origin, it gave off a branch which was of larger caliber and 1 cm. in circumference. This appeared to represent the right common iliac artery. On the left side, about 5 cm. from the bifurcation of the abdominal aorta, a second bifurcation occurred: one branch, which was 1 cm. in diameter, apparently represented the left common iliac artery, and the other, the mid-sacral artery. The latter was about 0.8 cm. in circumference. The hypogastric arteries were not identified. No anomalous veins were noted.

The left lung weighed 340 grams, and the right, 480 grams. The upper lobes were crepitant and flabby. The right middle lobe and the lower lobe were firmer in consistency, and many firm, nodular areas could be palpated beneath the pleura. On section, these were seen to extend into the parenchyma of the organ. They were fairly sharply circumscribed from the surrounding parenchyma and were of grayish to dark red color. Bloody fluid could be expressed when the lung was squeezed. On section of the lungs, the upper lobes were of grayish color and air-containing. The right middle lobe and both lower lobes were of a pinkish color.

The spleen weighed 180 grams. There was an old area of infarction which had been replaced by fibrous tissue. The organ was congested.

The liver weighed 1,210 grams. The edges were very sharp. There was a reddish mottling evident through the capsule. On section, the central areas were dark red and somewhat depressed, and were fairly well demarcated from the surrounding yellowish-tan portal areas. The organ was firm in consistency. Certain portal areas, especially near the surface, semed to be reduced in size, and there was a network-like pattern of a reddish color.

The left adrenal was of normal size and shape. The right adrenal was adherent to the diaphragm and was flat in outline. Its dimensions were 3 by 3 by 4 centimeters. On section, it appeared to be normal.

The right kidney was absent. The left kidney weighed 245 grams. It was of somewhat crescentic shape with the concavity directed toward the aorta. There were remnants of fetal lobulations. The calices, pelvis, and ureter were normal. There were two arteries supplying this kidney. The superior vessel appeared to be the chief source of supply and was about 0.5 cm. in diameter. A second vessel arising from the aorta, about 2 cm. below the first, was about 2 mm. in diameter.

Only the left half of the trigone of the urinary bladder was present. The prostate was normal.

On the right side, the vas deferans arose at the epididymis. About 4 cm. from its origin, there appeared to be occlusion of the lumen because the milky, fluid contents could not be squeezed beyond this point. The vas deferans coursed in the spermatic cord through the inguinal canal and, as it emerged from the internal inguinal ring, became adherent to the anomalous right common iliac artery previously described. It then doubled back upon itself, again merging with the spermatic cord until it approached the epididymis. At this point, it once more turned upon itself and coursed up the spermatic cord to terminate in an aberrant seminal vesicle, located on the right anterolateral pelvic brim in the normal position of the common iliac artery. The right seminal vesicle contained fluid which was dark reddish-green and which closely resembled that which might be found in a liquified hematoma. The left seminal vesicle appeared normal and contained the usual light tan fluid. The testicles were both in the scrotal sac, and were of normal size and consistency. The tubular architecture appeared normal.

The brain weighed 1,300 grams. On cutting the left lateral ventricle, a cloudy grayish fluid escaped. There was diffuse softening in the medial portion of the left cerebrum, just adjacent to the sagittal fissure. The vessels appeared to be normal. Upon sectioning the brain after fixation, the large area of necrotic softening on the left side was found to extend from the genu of the corpus callosum posteriorly to the tip of the occipital lobe, on its medial aspect. There was destruction of the basal ganglia, including the thalamus on the left side. There was extension from the ventricle out to the cortex of the temporal lobe. The right cerebrum and the cerebellum were intact.

Histologic Studies.—Sections of all organs were examined routinely. Only the significant ones are described.

The epicardium and endocardium of the heart were slightly thickened in some areas. The muscle fibers were larger than normal, and there was a great deal of hydropic degeneration. There were small areas of fibrosis scattered throughout the myocardium. There were old organized and recent thrombi adherent to the endocardium.

Sections from several areas of the aorta showed normal architecture. Measurements of fixed sections of the aortic wall, mounted on glass slides, indicated that the thickness varied from 0.1 to 0.15 centimeter.

The lungs showed scattered patches of bronchopneumonia with many large clumps of bacteria in some areas. One pulmonary vessel contained a thrombus. The vessel wall at this point was partially destroyed and was infiltrated with many acute cells and a few round cells. Surrounding this vessel, many of the alveoli contained red blood cells, and there were areas of bronchopneumonia in which the alveolar walls were partially broken down. Many of the alveoli contained large numbers of macrophages. There were many polymorphonuclear leucocytes in the bronchi.

The spleen showed increased trabeculation and congestion.

The liver showed some surface atrophy, and many of the central areas were greatly congested, with atrophy of the cord cells in these areas. There was some fat in the cells of the portal areas.

The right seminal vesicle had thick fibrous walls, and the glandular epithelium was poorly developed. The left seminal vesicle and prostate appeared to be normal.

In and beneath the cortex of the brain, there were areas of marked disintegration of tissue. The cerebral vessels were congested.

The anatomic diagnosis was: anomalous vascular and genitourinary systems; hypoplasia of aorta; congenital absence of the right kidney and ureter; anomalous right seminal vesicle and vas deferens; cardiac hypertrophy and dilatation; focal myocardial fibrosis; mural thrombi, right auricle and left ventricle; infarct of left cerebrum, recent; necrotizing bronchopneumonia; acute bronchitis; fibrinous pleural adhesions, bilateral; hydrothorax, bilateral; atelectasis; hydropericardium; hydroperitoneum; congestion of viscera; central necrosis of liver; fatty liver; jaundice; pulmonary embolus; compensatory hypertrophy of left kidney; old infarct of spleen; and emaciation.

COMMENT

This case falls into the general category of those in which cardiac insufficiency has been the result, solely, of a hypoplastic arterial system. The diagnostic feature was a youthful patient, previously vigorous and without cardiac symptoms, who developed cardiac enlargement and severe insufficiency with a progressively downhill course. The cardiac enlargement was nondescript, there were no murmurs heard, and no valvular or congenital cardiac abnormalities were found at the post-mortem table. The coronary arteries, themselves, were normal. It is unusual in that the presenting complaint was that of a hemiplegia, which developed over a period of a few days, and in the occurrence of extreme anomalies of the genitourinary system. Abnormalities of the genitalia are common in aortic hypoplasia, but such extensive anomalies of the genitourinary tract, as presented here, have not been previously recorded, to the authors' knowledge. At the post-mortem table, the exact cause of cerebral infarction could not be ascertained. From the mode of onset it was felt that cerebral thrombosis, probably secondary to a small cerebral embolus, was the most likely explanation.

It was generally agreed, by all who saw the patient, that diffuse vascular disease of some sort was present, and periarteritis nodosa was considered as a possibility. The correct diagnosis was suspected antemortem by virtue of (1) the absence of the aortic knob roentgenographically and the absence of an aortic indentation on the barium-filled esophagus by fluoroscopy, (2) the nondescript, diffuse type of cardiac enlargement, without murmurs or demonstrable valvular disease, occurring in a young man, and (3) the evidence of anomalies of the peripheral arterial tree, as indicated by examination of the vascular supply to the extremities.

Although not suspected during life, the congenital abnormalities of the genitourinary tract are of interest, not only because of their bizarre character, but also because they suggest that, in this case, at least, the aortic hypoplasia represents a congenital abnormality and not a developmental arrest.

It is desired to call attention to the fact that, while significant aortic hypoplasia is a rare finding, it should be given more consideration than has been accorded it in recent years by the medical profession, and that it can be the only demonstrable cause of severe and fatal heart disease in young adults.

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TRAUMATIC RUPTURE OF BOTH VENTRICLES OF THE HEART

CASE REPORT

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THE importance of traumatic heart disease is being made manifest in recent years by the increased number of publications and more frequent recognition of this clinical entity.[1-3] This problem is frequently encountered in industry from a medicolegal standpoint. It is the purpose of the paper to present a case of extreme cardiac injury associated with little evidence of external trauma.

CASE REPORT

A Negro male, aged 21 years, without history or evidence of previous cardiac disease, was brought to the hospital and found to be dead on arrival. It was reported that this man had been pinned between the cabin and chain cable of a small crane ditchdigger. The chest

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was compressed in an anteroposterior direction, at the level of the lower sternum (Fig. 1). Approximately four minutes elapsed before he could be freed.

Autopsy Report.—The necropsy was performed two hours after the accident. A small abrasion was present in the right infrascapular region over the eighth and ninth ribs, about three inches from the midline. There were also small depressions on the skin overlying the lower portion of the sternum just above the xyphoid process and extending to the left shoulder. The remainder of the remarkable findings were limited to within the abdomen and chest.

Examination revealed linear fractures of the right eighth and ninth ribs, two inches from their vertebral attachments. There was no evidence of injury to the underlying lung. The pericardial sac was markedly distended with a volume of liquid and clotted blood which

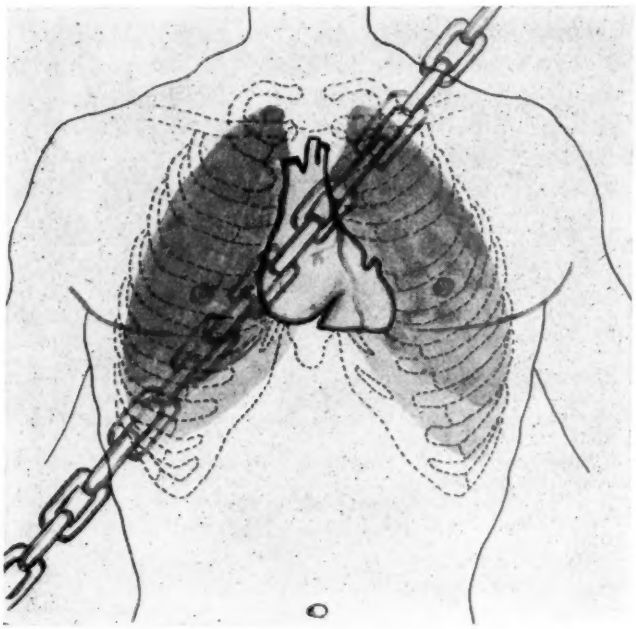

Fig. 1.—Schematic illustration showing location of compression force on chest resulting in rupture of the heart.

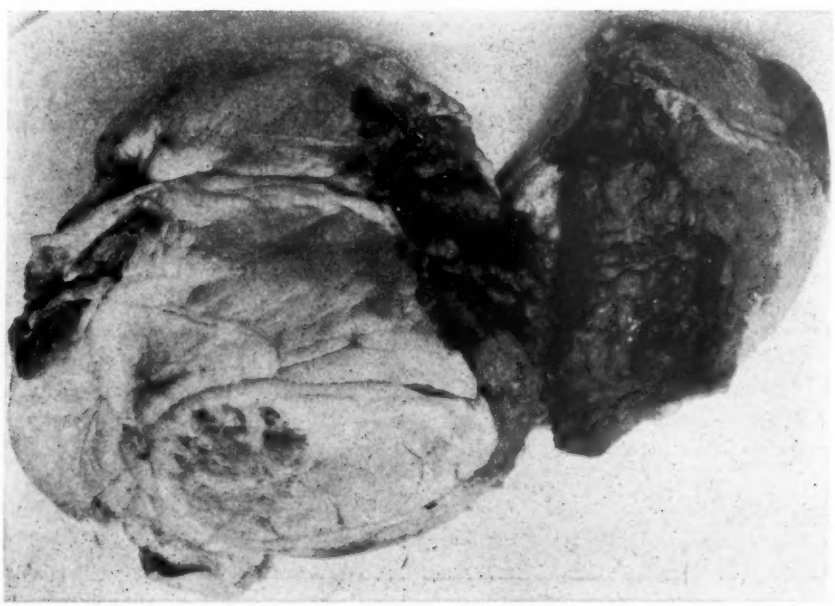

Fig. 2.—Traumatic rupture of both ventricles of the heart, posterior view.

measured 350 cubic centimeters. A large hematoma was seen protruding from the apex of the heart. A jagged tear, 11 cm. long, at the junction of the lower one-third and upper two-thirds of the posterior surface of the heart was present, which extended completely through both ventricles (Fig. 2).

On opening the abdominal cavity, 1½ liters of liquid and clotted blood were found. The liver was of average size and normal in appearance. On the posterior aspect of the right lobe of the liver there was an irregular laceration, approximately 3 cm. long and 3 cm. deep, which accounted for the intra-abdominal hemorrhage.

DISCUSSION

Traumatic heart disease has escaped recognition by many clinicians, chiefly because it was believed that the heart was well protected within the bony cage of the chest. The heart is particularly vulnerable because of its close approximation to the anterior chest wall. It has been found to suffer serious injuries from relatively minor trauma, both direct and indirect.

Beck, in 1935, made an exhaustive study of the mechanism causing traumatic heart disease.[3] More recently the literature has contained numerous articles illustrating how varied this condition may be.[2, 4, 5] Direct trauma to the anterior chest wall, such as that produced by a thrown ball, or a steering wheel, may produce contusion of the heart muscle. The resulting symptoms are not unlike those seen in acute myocardial infarction and may be accompanied by the corresponding physical signs and electrocardiographic changes. Patients suffering from contusion of the heart are likely to run a course similar to that in persons with acute coronary occlusion. Convalescence is usually uneventful but is, occasionally, complicated by myocardial failure. While spontaneous rupture is rare, it occurs most frequently in unrecognized cases during the second week following exertion.

Cardiac ruptures are also produced by broken ribs which are driven into the heart. Increased intracardiac pressure by application of compression forces to the legs and abdomen is capable of causing heart rupture.

A fourth mechanism causing bursting of the heart is that of compression between the sternum and vertebrae. A "blow out" results from the greatly increased intracardiac pressure, caused by constriction of the great vessels at the base of the heart. It is believed that this latter explanation is illustrated by this case report. The work of Bright has demonstrated the ease with which nonpenetrating wounds of this type may injure the heart muscle.[6]

World War II exposed large groups of persons to compression types of injuries. Ever increasing speed of transportation, both on the ground and in the air, will undoubtedly result in a greater incidence of traumatic heart disease.[5] The presence of more obvious injuries, such as fractures or lacerations, should not lead the physician to assume that the cardiovascular system has escaped damage.

CONCLUSIONS

1. A case report of traumatic rupture of both the right and left ventricles with minimal evidence of external injury is presented. Search of the literature fails to reveal a similar lesion of this extent.

2. Traumatic heart disease is not uncommon and is apparently being recognized with increasing frequency.

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ISOLATED MYOCARDITIS PROBABLY OF SULFONAMIDE ORIGIN

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ISOLATED myocarditis is a pathologic term indicating an inflammatory process localized in the myocardium to the exclusion of the pericardium, endocardium, and (in the opinion of some) other tissues of the body. At present, most generally accepted entities of myocarditis are excluded; therefore, the etiology of isolated myocarditis itself must be unknown. This disease most often begins with a chill followed by dyspnea, precordial distress, and weakness with a rapidly progressing myocardial failure of undeterminable cause, often culminating in sudden death (Magner,[1] Helwig and Wilhelmy,[2] and Major and Wahl[3]). Subacute and chronic forms lasting months are recognized by Simon and Walpaw.[4] This uncommon condition is rarely diagnosed before death.

Various etiological agents such as unknown viruses, peculiar hypersensitivities, vitamin deficiencies, and reactions to chemicals have all been suggested. Many of the cases presented to date have been associated with clinically classifiable inflammatory diseases including influenza, pneumonia, scarlet fever, typhoid, phlegmon of the foot, and gonorrheal urethritis. To include these cases in a classification of isolated myocarditis, one must assume that known bacteria and viruses did not cause the myocarditis or else the definition given above must be broadened. Animal experimentation[5] may be of aid in clarifying the probable variety of causes.

A considerably varied histopathologic picture has been described in the literature as identifying isolated myocarditis or one of its several synonyms (Fiedler's, primary interstitial, circumscribed, diffuse idiopathic, productive, pernicious, and eosinophilic myocarditis). The disease has been divided into two distinctly different types, the granulomatous and the diffuse. In the first, the histologic change might well be confused with certain cases of tuberculosis, syphilis, tularemia, and mycosis. The difference is that the exact etiology cannot be proved in isolated myocarditis. Further, the patient should not have any known specific disease elsewhere in the body which might cause a granulomatous change in the myocardium. In this type one would expect patchy isolated lesions with a prominent fibrous element and inflammatory cells, possibly including lymphocytes, plasma cells, eosinophiles, monocytes, and giant cells in varied proportion.

The second, and more frequent, diffuse variety of isolated myocarditis has been described by Covey[6] as showing a common microscopic pattern with minor variations and by Saphir[7] as being mimicked by so many known inflammatory

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diseases of the myocardium that it cannot be considered to have a characteristic histology. There is not only an interstitial but also a parenchymal reaction in some cases. The interstitial tissues are diffusely infiltrated by varying numbers of macrophages, lymphocytes, eosinophiles, and neutrophiles, and there are varied amounts of diffuse fibroblastic reaction. Changes in muscle fibers are frequently described in this type of isolated myocarditis. They include focal areas of swelling, necrosis, sarcolysis, myolysis, "granular decomposition," and "explosive necrobiotic" changes.

French and Weller[8] have reported 126 cases of interstitial myocarditis associated with sulfonamide therapy. They further reproduced an eosinophilic type of diffuse myocardial reaction in animals given various sulfonamides in dosages comparable to those given to human beings.

CASE REPORT

A 43-year-old man was first seen by one of us (S. G. S.) in his hotel room at about 3:00 P.M. on Oct. 23, 1943, five hours before his death. He stated that he had been ill with the "flu" for the past week or ten days. Just prior to his calling a physician he had been nauseated and had vomited profusely. The vomiting was not of a projectile type but was very copious. He suffered from a severe chill of malarial type which lasted for five minutes and he could not keep himself warm in spite of several extra blankets. In view of his extreme discomfort and condition, hospitalization was advised at once. He was admitted to St. Luke's Hospital at about 4:30 P.M. The physical examination, both at the hotel and the hospital, revealed the temperature to be 103.4° F. The pulse was 120 and the respirations were 24 per minute. The systolic blood pressure reading was 120 mm. and the diastolic was 84 mm. of mercury. His hair was sparse on the crown. The eyes, ears, nose, and throat were all essentially normal. Examination of the heart revealed no murmurs. Its rate was regular. The tones appeared to be of a distant type and seemed impaired in quality. There were no râles or other changes in the lung fields. No masses were palpated in the obese abdomen. There was no tenderness or rigidity. The rectal examination was negative. No edema of the extremities was noted. There was no urethral or prostatic discharge. The patient felt fairly comfortable after his hospitalization, and his only request was for sleep because he was completely fatigued.

A urinalysis revealed an amber urine with a pH of 5; specific gravity of 1.003; albumin, 4 plus; and sugar test, negative. Microscopic examination of the urine revealed 20 to 25 red blood cells and 8 to 10 white blood cells per high-power field. The hemoglobin was 93 per cent or 13.25 grams. The red blood cells numbered 4,620,000 per cubic millimeter; the white blood count was 12,400, with the following differential count: nonsegmented polymorphonuclears, 23 per cent; segmented polymorphonuclears, 62 per cent; and lymphocytes, 15 per cent.

Because of the very severe chill, indicating a possible blood stream infection, a blood culture was taken immediately after admission. After nineteen days of aerobic and anaerobic culture there was no growth. A definite diagnosis was not made on admission although it was assumed that the patient might be suffering from influenza, which was prevalent at that time, and probably also had a blood stream infection.

The patient revealed that, on October 5, he had had extramarital relations with a woman proved later to have gonorrhea. Because of this exposure he had been advised by a physician to take sulfadiazine. He had been taking 15 grains three or four times a day since October 14 (ten days), without medical supervision. There was no past history of any heart disease.

No medication was given the patient while in the hospital because it was the feeling that the sulfadiazine itself might possibly have contributed to his condition. Until a more definite diagnosis was made, therapy did not seem to be indicated. At about 8:00 P.M. on October 23, about four hours after admission, the nurses, in making routine rounds, found the patient dead.

Autopsy Findings.—This 43-year-old, well-developed and slightly obese white man measured 67 inches in height and weighed about 200 pounds. The only outstanding gross pathologic change was in the heart, which weighed 460 grams. This increase in size was due mainly to enlargement of the left ventricle. However, there was a mild dilatation and slight hypertrophy of the right ventricle. The left ventricular cavity was moderately dilated.

Its walls were remarkably pale and somewhat mottled throughout. It was not of the usual flabby consistency of a severely degenerated myocardium but maintained its form fairly well and was not friable. The trabeculae carneae in both ventricles were fairly prominent. A careful search revealed no myocardial fibrosis, and there was no significant sclerosis of the three principal coronary arteries or their visible main branches. The auricular cavities were normal in size, and their endothelial linings were transparent. The four valves had no fibrosis or vegetations and were apparently competent. The pericardium was transparent and free from injection and adhesions.

Fig. 1.

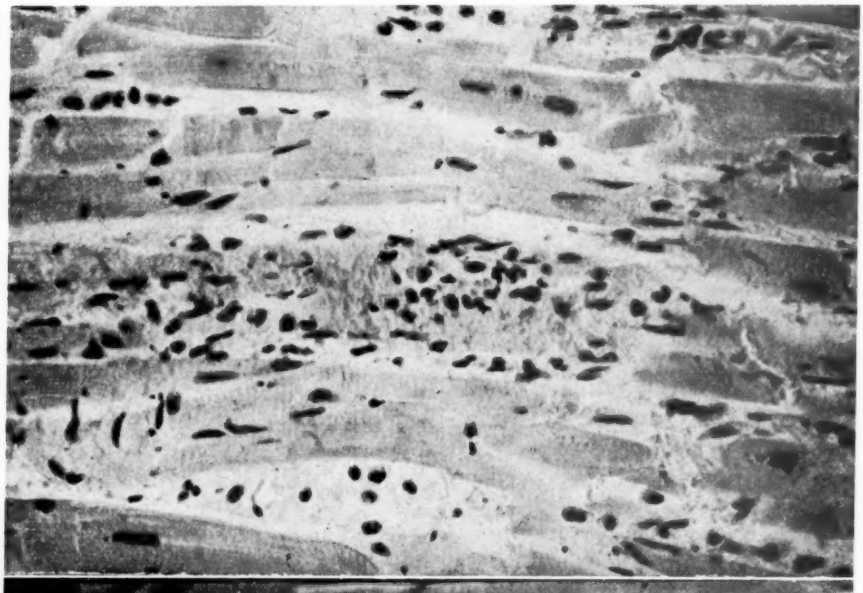

Fig. 2.

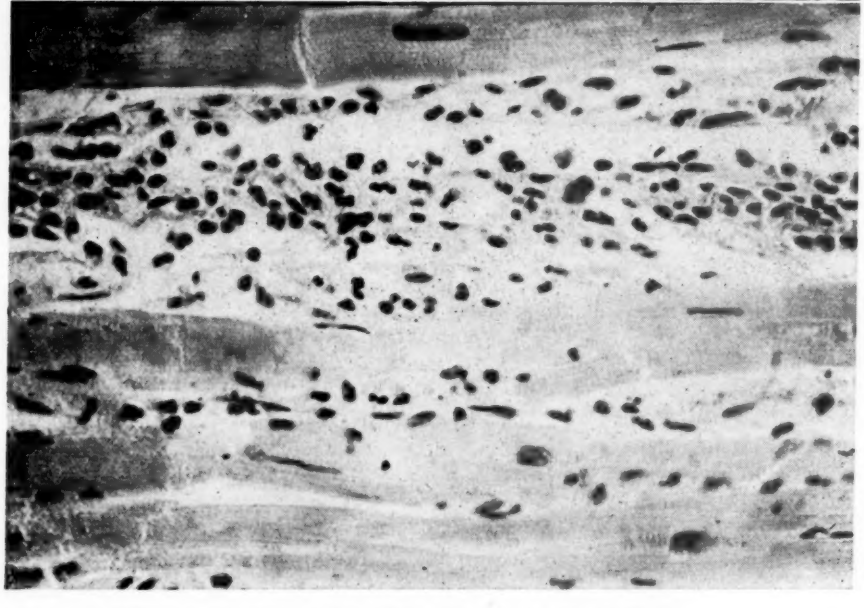

Fig. 1.—Diffuse infiltration of interstitial tissues of myocardium.
Fig. 2.—Swelling and granular necrosis of muscle fiber with neutrophilic reaction.

The other gross findings might well have been entirely secondary to a severe inflammatory disease. There was a moderate congestion and edema of the dependent portions of both lungs without gross evidence of chronic passive congestion or pleural fluid. The liver was somewhat pale, mottled, and flabby. It weighed 2,120 grams. The spleen, which weighed

320 grams, had an extremely soft, friable, granular, dark-red, cut surface. The kidneys, together, weighed 380 grams. Their parenchymal tissue was pale and slightly swollen throughout. The brain weighed 1,430 grams and appeared to have displaced much of the cerebral spinal fluid.

The microscopic study of the heart revealed an acute diffuse type of inflammatory process (Fig. 1), characterized mainly by an interstitial reaction with a predominance of neutrophiles in moderate numbers, swollen endothelial cells and fibroblasts in slightly increased numbers, and occasional eosinophiles, monocytic phagocytes, and rare lymphocytes. There were also diffuse parenchymal changes, characterized by patchy irregularities in the staining of the muscle fibers, with occasional isolated, faintly granular changes in the muscle fibers. Rarely did one find a severely swollen segment (Fig. 2) with coarse, deeply acidophilic granules and beginning fragmentation. A neutrophilic reaction about these areas was noted. There were no Aschoff's bodies, and the reaction was no more severe about the smaller arteries than it was elsewhere in the interstitial tissue. The smaller arteries had no significant thickening of their walls. Special stains for bacteria failed to disclose their presence.

Moderate secondary toxic changes were evident in the liver, spleen, and kidneys. There was a mild, but distinct, increase in lymphocytic and monocytic infiltration in the portal areas of the liver. No significant inflammatory cell reaction was found in the lungs; however, there was a diffuse congestion and edema.

DISCUSSION

The histologic changes in the myocardium in the case presented are typical of those described as a diffuse form of isolated myocarditis. We are much inclined to believe that they were due to the preceding sulfonamide therapy. However, influenza, gonorrhea (neither of which was proved to have been present), or unknown causes cannot be ruled out.

In cases previously reported of fatalities following sulfonamide medication, the question arises as to what part the primary condition, for which the chemotherapy was prescribed, may have contributed to the fatality itself as well as to the histologic changes. The presence of eosinophiles in the histologic picture in the myocardium suggests the possibility of hypersensitivity to the drug. This individual, unfortunately, continued to take the sulfadiazine as a prophylaxis for gonorrhea, which was never proved to have existed. The symptoms he suffered from can be well explained by the somatic deterioration from hypersensitivity to the sulfonamide. It is interesting, indeed, to note that, on questioning, no oliguria or anuria was reported by the patient. The microscopic examination of kidneys revealed no evidence of the usual kidney complications with deposition of sulfonamide concretions.

A brief discussion of the practical problems in the classification of myocarditis, in general, and isolated myocarditis, in particular, is pertinent. There are few pathologic terms in medicine used more loosely than myocarditis. Its use in connection with coronary sclerosis and the resulting complications has been gradually disappearing, almost to the vanishing point in current medical literature. However, it remains in frequent use among physicians in general. A greater problem at present is its use in connection with a variety of inflammatory, metabolic, degenerative, malignant, and deficiency diseases in which the clinician finds definite evidence of myocardial inadequacy complicating some known disease. In many of these disease processes the possibility of true myocarditis may exist. It is extremely difficult and often impossible to distinguish clinically between the far more common degenerative processes of the myocardium and the rare, truly inflammatory lesions. As a matter of fact, in some specific instances histopathologists are not entirely agreed as to when a "degenerative" process ends and an inflammatory reaction begins. Generally speaking, the presence of inflammatory cells, including the white cells of the blood, and the inflammatory cells developing from local tissue cells, are necessary

in the definition of an inflammatory process in any tissue of the body. There is undoubtedly a definite trend among clinicians to limit the use of the diagnosis "myocarditis" to conditions in which there is a histologically demonstrable inflammation in contradistinction to a degenerative change or a vascular disease of the myocardium.

The rarity of, and, to a greater extent, the lack of interest in, isolated myocarditis undoubtedly accounts for the present unsatisfactory understanding of the classification. There is good reason to believe that there are a variety of causes for the cases now published under this name. Therefore, it should be used purely as a pathologic term and not as the name of a disease entity. As each new cause for the myocarditis is proved it should be given a separate classification such as influenzal myocarditis or possibly arsphenamine myocarditis.

CONCLUSIONS

1. A case of diffuse isolated myocarditis is presented in which sulfadiazine may well have been the etiological agent.

2. An acceptable, clear-cut definition of isolated myocarditis cannot be arrived at after a review of the reported cases in the literature.

3. The name should be applied only to cases in which the etiological agent is unknown.

4. With an increased knowledge of the condition, it is likely that several disease entities may be distinguished.

5. The present widespread misuse of the term "myocarditis" is the result of long standing carelessness. It is a difficult clinical diagnostic problem to differentiate inflammatory from degenerative and vascular changes in the myocardium.

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MYOCARDIAL INFARCTION SUPERIMPOSED ON SHORT P-R, PROLONGED QRS COMPLEX

A Case Report

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SINCE a search through the available medical literature failed to show any report of a case of "short P-R, wide QRS complex" complicated by myocardial infarction, it was thought worth-while recording such an incident.

CASE REPORT

A 41-year-old soldier with six months' service in the Army was admitted to the Station Hospital on March 1, 1943, because of a sudden episode of smothering and choking, followed by severe pain over the lower sternum, which had occurred on Feb. 28, 1943.

The patient had always been active in the past, working as a cook and miner. The family history was noncontributory. His past history was negative except for the fact that he had frequent paroxysms of rapid heart action, and two previous attacks of precordial pain with numbness radiating to both arms in June, 1942. These latter episodes lasted only a few minutes and caused the patient to remain home for a few days. This pain was similar in character to the one which caused him to seek admission to the hospital. There was no history of previous dyspnea or peripheral edema. His paroxysms of tachycardia were usually short in duration, would occur sometimes at rest, and were never incapacitating.

The patient felt quite well until February 28, when, while wrestling with some companions in his hutment, he suddenly felt "choked and smothered" and experienced severe pain over the lower sternum and epigastrium. He lost consciousness and later felt nauseated and had a "soreness" throughout his chest. He remained in bed most of the day, getting up, however, to eat dinner. He slept poorly that night. The next day he became extremely nauseated, felt very weak, but had no pain in the chest.

On admission, the patient did not appear to be in marked distress. There was no cyanosis. The thorax was emphysematous in type, and the lungs showed a few fine râles in the right anterior thorax from the fourth intercostal space to the costal margin. These findings were not constant. The heart was not enlarged to percussion. The sounds were of good quality with rate slow and regular. The aortic second sound was equal in intensity to the pulmonic second. No murmurs were heard. The blood pressure was 120/90. A small abdominal hernia was noted in the midline between the umbilicus and xiphoid process, but there were no adherent intestinal contents. Soon after arrival at the hospital, he had one episode of difficult breathing. An electrocardiogram was taken that morning.

The patient was kept in bed, given phenobarbital and aminophylline and frequent small feedings. The temperature rose on the second day to 99.8° F. by mouth. The blood pressure ranged from 110/60, on the day of admission, to 100/70 on the fifth day and remained at this level thereafter. He felt well all the time except for an occasional tight feeling in his chest. The lungs cleared completely, and at no time did he show evidence of congestive failure. The heart sounds remained of good quality with a slow regular rate. He had no episode of paroxysmal tachycardia or cardiac arrhythmia at any time. Occasionally a faint systolic murmur was heard at the apex, but a friction rub was never noted.

The urine showed no abnormalities. The initial leucocyte count, taken soon after admission, was 5,200 per cubic millimeter with 67 per cent polymorphonuclears and 33 per cent lymphocytes. The hemoglobin was 85 per cent. The sedimentation rate was 14 mm. in one hour. Ten days later the white blood cell count rose to 11,650 per cubic millimeter; the sedimentation rate remained at 14 millimeters. On the thirty-ninth day after admission, the white blood cell count was 18,600 and the sedimentation rate was 6. An x-ray examination of the chest showed the heart and lungs to be normal.

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Electrocardiograms, taken on the day of admission, the third day, and the fourteenth day (Fig. 1), suggested a posterior wall infarct superimposed on a prolonged QRS, short P-R interval of the "Wolff-Parkinson-White syndrome."

About six weeks after admission an exercise test was performed with the patient going up and down a three-step stairway while holding an ice cube in his right hand. After eighteen trips he complained of a pressing and aching sensation over the precordium which lasted thirty seconds. This pain was similar in nature to that which had caused his admission but was shorter in duration. There was no radiation of this pain. About one week later, this test was repeated under similar circumstances and another episode of precordial aching was precipitated after twenty-two trips. This time the pain lasted twenty seconds. On the same day the test was repeated without the ice and the patient performed thirty-four trips before experiencing the same pain.

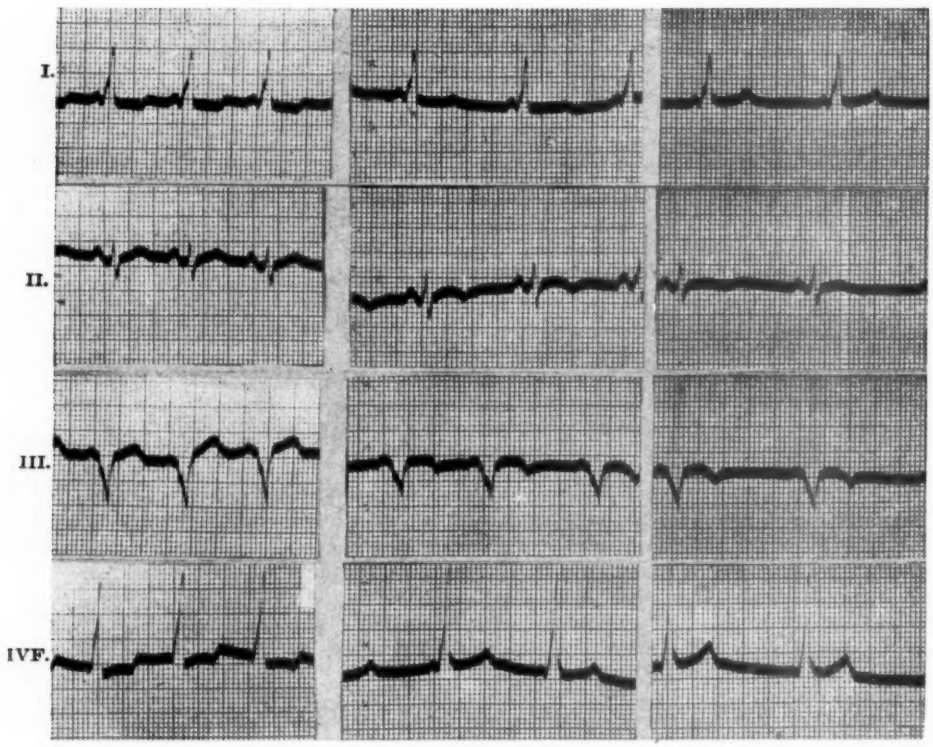

Fig. 1.—The first column of tracings on the left was taken on the day of admission: rate, 96; P-R interval, 0.09 second; QRS interval 0.11 second with slurring of upstroke of R_1 and downstroke of Q_3; left axis deviation; prominent S_2; T_1 diphasic; T_2 and T_3 upright and T_4 diphasic with S-T depression of 2 millimeters. The second column of tracings was taken three days later: T_1 upright, small amplitude; inversion of T_2 and T_3. These marked T-wave changes suggested a posterior wall infarct, superimposed on short P-R, prolonged QRS interval of the "Wolff-Parkinson-White syndrome." The third column of tracings was taken two weeks later: T_2 had become upright, low amplitude; persistent cove-shaped inversion of T_3. Wolff-Parkinson-White findings persisted unchanged.

Just prior to these exercise tests the white blood cell count was 11,350 per cubic millimeter, with 6 per cent stab cells, 63 per cent segmented forms, 29 per cent lymphocytes, and 2 per cent eosinophiles. About a week after the last exercise test the white blood cell count was 9,850, and the sedimentation rate was 16 mm. in one hour. The following week the sedimentation rate was found to be 20 mm. in one hour. Just prior to discharge the white cell count was 7,750, and the sedimentation rate had come down to normal.

The course of the electrocardiographic tracings during this period is recorded below (Fig. 2).

Two and one-half months after admission the exercise test was again repeated while the patient held an ice cube in the right hand. After forty-five trips precordial pain was reproduced which lasted fifty seconds. Precordial tracings which were taken immediately before and after the last test showed a marked inversion of the T wave but no deviation of the S-T segment during the pain (Fig. 3).

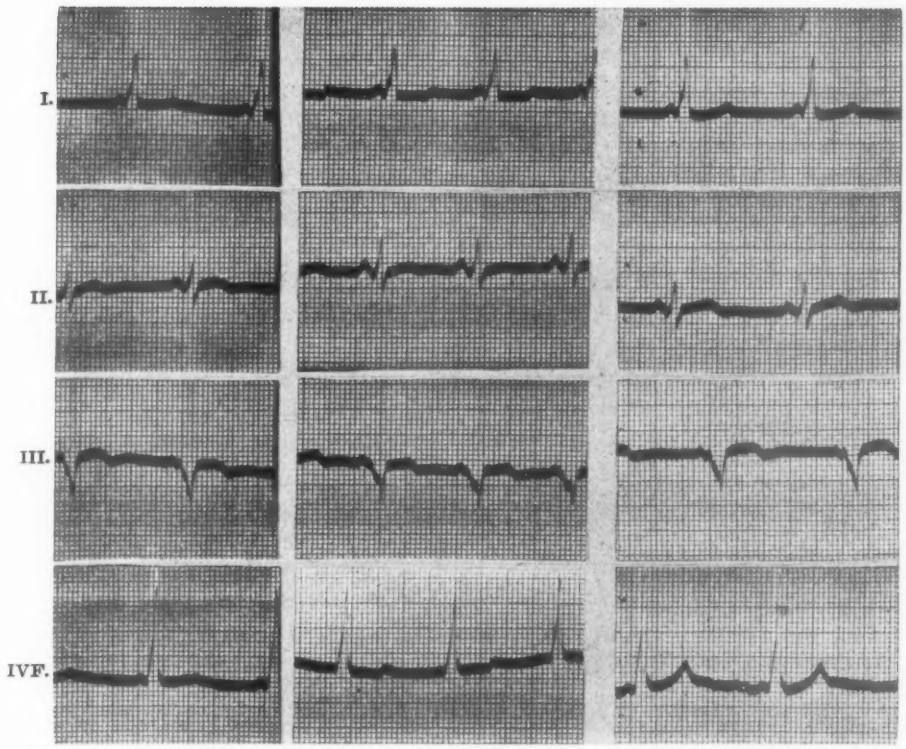

Fig. 2.—The first column of tracings on the left was taken one month later: little change over previous tracing except for slightly greater amplitude of T_3. The second column of tracings was taken nine days later: T_1, which had previously been upright and of low amplitude, diphasic; T_{AVF} diphasic; T_2 and T_3 upright. The third column of tracings was taken about three weeks later: T_1, T_2, T_{AVF}, upright; T_3 diphasic with coving. T_{AVF} shows sharp upright pickup.

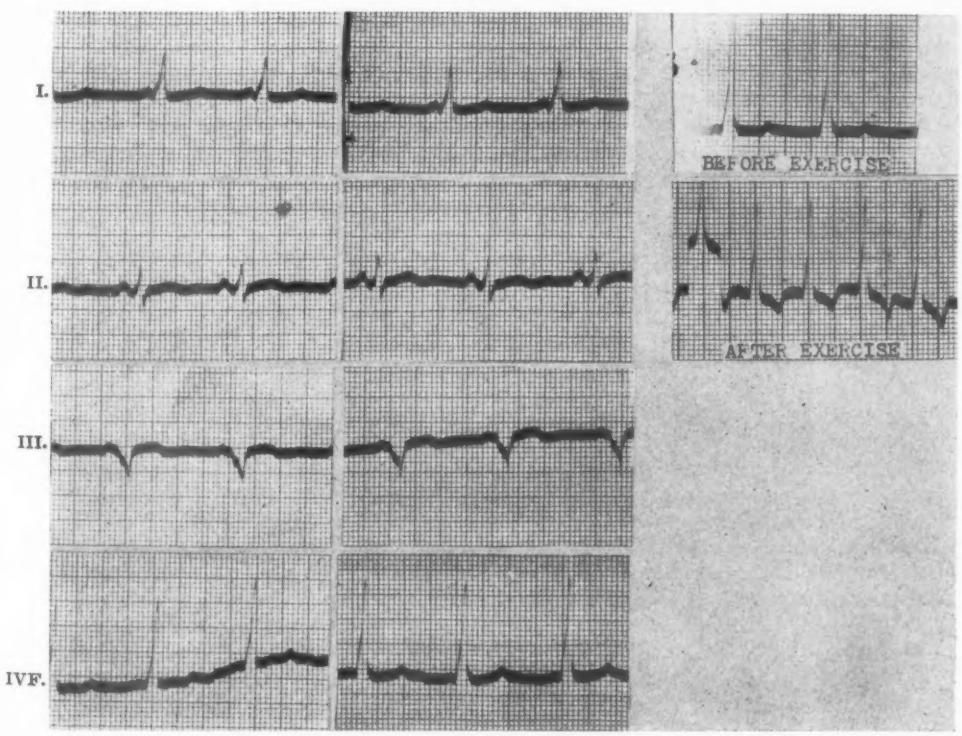

Fig. 3.—The first column of tracings on the left was taken two months after admission: inversion of T_3 less marked. The second column of tracings was taken one week later: no significant changes. In all these tracings there was a short P-R interval, widened QRS with slurred R_1, and deep notched Q_3. The third column of tracings was taken one week later: (Forty-five trips produced precordial pain lasting fifty seconds). Upper strip before test showed precordial tracing to be normal. Immediately after exercise lower precordial strip showed marked inversion of T wave, but no deviation of S-T segment during pain.

COMMENT

A consideration of the significance of the findings in this case resolves itself into two possibilities: 1. Is this short P-R interval, wide QRS complex, part of the electrocardiographic picture of myocardial infarction? 2. Is this a case of myocardial infarction in a person who had exhibited previously the Wolff-Parkinson-White syndrome?

For absolute proof of myocardial infarction, superimposed on a short P-R, wide QRS complex, there would have to be cardiographic evidence that this complex existed prior to the episode of myocardial infarction. The evidence does not exist in this case. However, the history of frequent attacks of paroxysmal tachycardia, along with the electrocardiographic changes, suggests that the Wolff-Parkinson-White syndrome was present prior to the coronary occlusion. Furthermore, we cannot find any literature on experimental or clinical bundle branch block or coronary occlusion with myocardial infarction in which the pathogenesis included shortening of the P-R interval along with the widening of the QRS complex.

It is concluded, therefore, that this patient probably represents a case of posterior wall infarction superimposed on the congenital anomaly of a short P-R interval and prolonged QRS.

Letters

To the Editor:

I have just read the article by J. A. Boone in the June, 1945, number of the AMERICAN HEART JOURNAL, page 751, entitled "Ventricular Fibrillation as a Complication of Hyperthyroidism."

It is my impression that a serious misinterpretation of the electrocardiogram has been made. Dr. Boone stated that the "Electrocardiogram (Fig. 1) showed auricular flutter, A-V dissociation, and intraventricular block. Lead II appears to consist entirely of a period of ventricular fibrillation." The article is titled and based upon this interpretation.

Clinically the patient presents the usual signs of auricular fibrillation—"rapid tumultuous heart action, grossly irregular, and the rate was 150 to 160 . . . the pulse deficit was 30 to 40." And the auricular fibrillation was of the paroxysmal type, since there was prompt restoration of the normal rhythm.

Leads I and III of the electrocardiogram show the characteristic findings of auricular fibrillation, namely absolute irregularity with irregular spacings of the ventricular complexes. There are here and there, small additional waves, which may be attributed to auricular activity (ff waves).

If these ff waves were due to auricular flutter, such waves, or accentuations of other waves, ought to appear regularly and periodically. They do not do so. The only real evidence for flutter is lacking.

If A-V dissociation were present, one would expect a ventricle beating without auricular control and, therefore, beating regularly. This is not so.

Lead II has been interpreted as ventricular fibrillation, and standing alone, it certainly "appears" to represent it; but standing between Leads I and III, it represents merely another aspect of rapid fibrillation. R_1 varies from 5 to 8 mm. in height. QS_3 varies from 3.5 to 5.5 mm. in depth. The algebraic sum (R_2) may vary considerably but can easily give some of the small and larger waves present in Lead II. The sum of the T_1's and T_3's, where the T_3's are more greatly upright than the T_1's are inverted, will also give positive waves. Fibrillary (ff) waves will explain the rest and add to the variations in height. It would be impossible to say which letter in Lead II is applicable to the different waves, but the diagnosis of *auricular* fibrillation is clear enough.

The condition present is the not infrequent one, in hyperthyroidism, of paroxysmal auricular fibrillation. It is not proper to allow the misconception implied in the title of this article to creep into the literature without correction.

(Signed)

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224 Physicians and Surgeons Bldg.
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Aug. 28, 1945

To the Editor:

I should like to comment on the article, "Ventricular Fibrillation as a Complication of Hyperthyroidism," by J. A. Boone, which appeared in the AM. HEART J. 29: 751 (June), 1945.

In my opinion there can be no doubt that the electrocardiogram reproduced in Fig. 1, upon which the whole report is based, shows auricular flutter (rate 376) with varying A-V conduction and intraventricular block. The same mechanism is present in all leads.

Yet, the author interprets Lead II as ventricular fibrillation and calls it the "first proved instance of ventricular fibrillation in hyperthyroidism to be reported." Some more fundamental mistakes are revealed by the author's interpretation of Leads I and III of Fig. 1 as "auricular flutter, A-V dissociation and A-V nodal tachycardia with intraventricular conduction delay."

I think it is regrettable that this case of "ventricular fibrillation in hyperthyroidism" will now find its way into the Index Medicus and will be quoted in the literature.

(Signed)

RICHARD LANGENDORF, M.D.

104 South Michigan Avenue
Chicago, Ill.
Sept. 11, 1945

Abstracts and Reviews

Selected Abstracts

Sulzer, R., and Duchosal, P. W.: Principle of Cardiovectography, Cardiologia 6: 236, 1942.

The Braun valves enable the combination of two linear vertical conductors and so the tracing of the horizontal projection of the electrical heart Vektor. The figures so obtained were called *Planogramme* (PG) by us. (Synonyms: vektodiagram, monocardiogram, triogram, etc.)

Owing to difference in form and conductibility of the tissue, the heart potentials in the human body are unequally distributed, wherefore, the law of projections, underlying the Einthoven triangle, is not quite perfect. The errors resulting therefrom are particularly apparent in planography. The present paper examines the matter of the distribution of the potentials in the human body for the purpose of drawing conclusions therefrom for the planogrammes and their interpretation.

These researches show that one of the most advantageous conductors consists in selecting for the horizontal conductors a plane situated below the heart. The distance, which guarantees agreement of the straight and oblique PG, stipulates that this plane should be situated in the region of the cristae iliacae.

The polarity of the conductors in planography and the manner of the connection of the Braun values are discussed. AUTHORS.

Mannheimer, E.: Calibrated Phonocardiography, Cardiologia 6: 281, 1942.

A new method, calibrated phonocardiography, is demonstrated and its value as clinical routine work is emphasized. AUTHOR.

Kartagener, M.: The Significance of Heredity for the Origin of Acquired Heart Disease, Cardiologia 6: 314, 1942.

The presence of coronary thrombosis was definitely observed once in two generations (mother and son, very similar electrocardiograms) and once in one generation, in two brothers in the early fifties. The one brother of the latter pair had at the same time a Buerger disease of the arteries of the extremities, which points to the possibility of the presence of arteriitis stenosans coronariae (von Albertini) as the cause of the coronary thrombosis. In a further observation, father and son, coronary thrombosis could be assumed with great probability.

The observation of sclerotic aorta insufficiency in two brothers, simultaneously suffering from kidney and bladder stone, is reported for the first time. AUTHOR.

Moia, B., and Acevedo, H. J.: The Electrocardiographic Diagnosis of Myocardial Infarction Complicated by Branch Block, Rev. argent. de cardiol. 11: 341, 1945.

Contrary to the opinion of many outstanding authors it is stated that in most cases of coexistence of bundle branch block and myocardial infarct, the electrocardiogram permits an easy recognition of the existence of the latter.

The diagnosis of posterior infarcts is made exclusively by the changes observed in the standard leads; in anterior infarcts by these and by changes in the precordial leads—sufficing in general C_4 and C_2 not being necessary to take multiple precordial leads.

In the standard leads the fundamental changes are seen in the S-T segment and T wave: their displacement instead of occurring in the same direction which is opposite to that of the greater wave of the QRS complex—as is characteristic of bundle branch block—occurs in alternative sense: if the S-T segment is elevated the T wave is inverted, and vice versa.

Furthermore, the character and direction of the convexity of the S-T segment are frequently inverted. Nonfundamental changes are the occurrence of Q_1 in left bundle branch blocks and Q_2 and Q_3 in right bundle branch blocks, as well as the appearance of M or W complexes of low voltage in DIII.

In precordial leads the fundamental change is the occurrence of a deep Q in the left leads, and the nonfundamental changes the elevation of the S-T segment and the T wave negativity, both of which are only observed in anterior infarcts.

Changes of the S-T segment and T wave disappear ordinarily in from fifteen to thirty days, those of the QRS complex, when present, are permanent allowing a retrospective diagnosis.

The practical importance of the diagnosis of the coexistence of bundle branch block and infarct is emphasized, especially so when the infarct is not accompanied by a typical clinical picture. AUTHORS.

Neurath, O.: Is Digitalis Indicated in Myocardial Infarction? J. A. M. A. 128: 1016, 1945.

Digitalis alone used in congestive failure occurring with auricular fibrillation in myocardial infarction in thirty-two patients at the Los Angeles County General Hospital proved to be more harmful than beneficial. The hazard ordinarily considered the greatest, that is, the production of fatal ectopic ventricular rhythm with sudden death, was not in evidence. There was no increased hazard from cardiac rupture. The mortality was increased largely by the production of fatal emboli to the greater circulation. On the basis of these data, digitalis administered alone for the congestive failure associated with auricular fibrillation and myocardial infarction would seem contraindicated. AUTHOR.

Rosenblatt, P., and Loewe, L.: Healed Subacute Bacterial Endocarditis, Arch. Int. Med. 76: 1, 1945.

Two cases of healed subacute bacterial endocarditis were encountered. Death in both instances was due to cardiac failure incident to aortic valvular insufficiency. AUTHORS.

Homburger, F.: Effect of Sodium Salicylate on the Sedimentation Rate of Erythrocytes in Vitro, Am. J. M. Sc. 210: 168, 1945.

Sodium salicylate in vitro causes a marked reduction of the sedimentation rate of erythrocytes, particularly if it is accelerated. In fresh plasma this effect takes place at salicylate levels of about 90 mg. per 100 milliliters. In plasma which has been kept at room temperature for twenty-four hours, the effect takes place at levels of 25 to 30 mg. of salicylate per 100 ml. of plasma. When salicylate is left in contact with fresh plasma for the same length of time, the slowing of the sedimentation rate occurs at low levels. The standing of plasma at room temperature has an insignificant effect on the sedimentation rate of fresh red blood corpuscles in such plasma.

No demonstrable changes of plasma fibrinogen of red cells are caused by sodium salicylate and changes of the pH in the plasma do not account for the effect of sodium salicylate on the sedimentation rate. The effect is inherent in the salicylate radical, as sodium benzoate and sodium bicarbonate are ineffective.

It seems possible that this property of sodium salicylate, demonstrable in vitro, may partly account for the remarkable slowing of the sedimentation rate seen in some patients who receive salicylates. AUTHOR.

Harris, T. N.: The Erythrocyte Sedimentation Rate in Rheumatic Fever, Its Significance in Adolescent and Overweight Children, Am. J. M. Sc. 210: 173, 1945.

Four hundred children, 215 of whom were girls, were observed during their convalescence from episodes of rheumatic carditis. In nine of these patients, eight girls and one boy, ranging from 9 to 16 years of age, the erythrocyte sedimentation rate remained persistently elevated long after the other signs, symptoms, and tests for active rheumatic infection had returned to normal or become stationary. These patients were ultimately allowed out of bed while the erythrocyte sedimentation rate was still elevated. Continued clinical observation followed the resumption of physical activity failed to reveal any indication that these children had been allowed out of bed prematurely.

Some theoretical and clinical implications of these data are discussed. AUTHOR.

Levy, R. L., White, P. D., Stroud, W. D., and Hillman, C. C.: Transient Hypertension, J. A. M. A. **128:** 1059, 1945.

In an earlier paper, based on an analysis of the medical records of 22,741 officers of the United States Army, it was shown that in the group with transient hypertension there was a greater incidence of later sustained hypertension and there were higher rates for retirement and for death with cardiovascular-renal diseases. Using the same material and indexes, the present study was designed to demonstrate possible differences in the prognostic importance of various systolic and diastolic levels of transient hypertension.

With respect to the later development of sustained hypertension and retirement with cardiovascular-renal diseases, all levels of transient hypertension, both systolic and diastolic, were significant. Of particular interest was the observation that slight degrees of elevation were important, even when the systolic level alone was involved. Of the greatest significance was a transient rise in diastolic pressure above 100 mm., especially as an early sign of subsequent sustained hypertension.

No significant differences were apparent between the various degrees of transient hypertension in relation to the death rates with cardiovascular-renal diseases. So, as is true for many other prognostic criteria applied to these conditions, the height of a temporary rise in blood pressure does not appear to foretell the severity or extent of the lesions which eventually are a cause of death.

The data given lend support to the view that transient elevations of blood pressure, above the upper range of normal, often represent an early stage of hypertensive vascular disease. AUTHORS.

Rundles, W. R.: Hemorrhagic Telangiectasia With Pulmonary Artery Aneurysm. Am. J. M. Sc. **210:** 76, 1945.

A case is reported of a patient with hemorrhagic telangiectasia who had suffered from repeated epistaxis from the age of 14 years, and in later life from gastrointestinal hemorrhage severe enough to produce a severe, incapacitating anemia. Multiple gastric telangiectasia were seen by gastroscopic examination. An aneurysm of the pulmonary artery was present which did not increase appreciably in size during a period of over seven years' observation. AUTHOR.

Mason, J. M., III, and Giddings, W. P.: Experience With Lumbar Sympathetic Ganglionectomy for Wounds of Major Blood Vessels of the Lower Extremity. Surg., Gynec. & Obst. **81:** 169, 1945.

Lumbar ganglionectomy, if it can be performed early, should be considered in the management of wounds from the bifurcation of the aorta to the bifurcation of the popliteal artery which jeopardize the circulation to the lower extremity. The real test may lie in its use in cases of severed popliteal artery, for very few extremities survive with injury to this vessel. Presence of an intact profunda femoris artery in the face of loss of the superficial femoral has been found to be no guarantee of the viability of the extremity. Collateral circulation of the lower leg has been found to be sufficient even in the presence of ligation of two of the three major vessels. It is felt that ganglionectomy will be less frequently indicated in injuries to vessels of the lower leg than in those of the thigh. AUTHORS.

Lawson, H., and Rehm, W. S.: The Reversibility of the Cardiovascular Damage Done by Nearly Complete Exsanguination, Am. J. Physiol. **144:** 206, 1945.

Barbitalized, moderately dehydrated dogs were almost completely exsanguinated by controlled bleeding in order to determine the liminal circulating blood volume. There appears to be a nearly straight-line relationship between the circulating blood volume in the intact unbled animal, and the liminal circulating volume. When the animals were kept alive by reinjection of blood, the liminal circulating volume as determined by a second, complete exsanguination was usually considerably elevated. The circulating plasma volume at the start of the final bleeding was usually less than the sum of the volume remaining at the end of the first plus the volume reinjected, by an amount which is nearly equal to the volume of extravascular fluid added during the first bleeding. It thus appears that nearly all the fluid which enters the circulation during the first bleeding leaves the circulation after the reinjection. If allowance was made for a systematic error in the measurement of circulating cell volumes, the cell volume at the beginning of the final bleeding appeared to be nearly equal to the sum of

the residual and the reinjected volumes. No reinjected cells thus appear to be withdrawn from the circulation. The average volume of blood drawn on the final bleeding was 9-7 c.c. per kilogram less than the sum of the residual bleeding volume, as estimated from control studies, and the volume reinjected. The elevation of the liminal circulating volume is just about enough to account for this loss of bleeding volume.

In similar experiments on dogs pretreated by intravenous injection of 15 c.c. per kilogram of 0.9 per cent sodium chloride solution, there was no evaluation of the liminal circulating volume of the final bleeding. The average volume of blood drawn on the final bleeding was only 4.4 c.c. per kilogram less than the sum of the residual bleeding volume and the volume reinjected. This moderate loss of bleeding volume following the reinjection appears to be due to a decline in circulating blood volume during the interval, and to a reduction in ability to replenish volume on the final bleeding. These changes cannot be attributed with assurance to any damaging effects of the first exsanguination, since the loss of bleeding volume in pretreated dogs is equally great if the first bleeding is terminated long before the exsanguination is complete. In dehydrated dogs the loss of bleeding volume is much less when the first bleeding is terminated earlier. AUTHORS.

Lawson, H., and Rehm, W. S.: The Efficacy of Gelatin Solutions and Other Cell-Free Fluids in Reversing the Effects of Nearly Complete Exsanguination, Am. J. Physiol. **144:** 217, 1945.

Barbitalized dogs pretreated by intravenous saline injection were almost completely exsanguinated by controlled bleeding and injected with various replacement fluids. The fatal hemorrhage volume four hours later was used as an index to the relative effectiveness of the fluids in reversing the effects of the first hemorrhage. As measured in this manner, the order of effectiveness of the fluids studied was: whole blood 3.9 per cent gelatin P-20 plasma and serum 3.45 per cent gelatin L-80 2.8 per cent gelatin B-20610-51 0.9 per cent sodium chloride. The gelatin solutions are approximately iso-osmolar, and have about the same colloidal osmotic pressure in vitro as dog plasma.

Dehydrated dogs (without saline pretreatment) were studied in similar experiments for evidence of damaging effects of gelatin P-20. The volume remaining in the circulation at death was not significantly greater than in dogs replaced with whole blood. Blood replenishment during the final hemorrhage was at least as great as in animals replaced with whole blood. The circulating blood volume maintained following the replacement was at least as great as that maintained by dogs replaced with whole blood. Replacement with higher concentrations of gelatin P-20 (6 per cent) produced larger circulating blood volumes. No evidence was obtained that the higher concentrations produced additional cardiovascular damage under these experimental conditions. AUTHORS.

Frank, H. A., Seligman, A. M., and Fine, J.: Traumatic Shock, X. The Treatment of Hemorrhagic Shock Irreversible to Replacement of Blood Volume Deficiency, J. Clin. Investigation **24:** 435, 1945.

The therapeutic value of various agents for the treatment of hemorrhagic shock, which is not responsible to the replacement of all shed blood, was tested under conditions calculated to avoid or to minimize the confusing effects of anesthesia, blood sampling, operative manipulations, and other forms of trauma. Utilizing the relatively simplified set of conditions described the following results were observed with the agents tested:

Massive infusions of saline may cause transitory improvement in circulation but do not cure hemorrhagic shock irreversible to transfusion.

Massive infusions of isotonic bovine albumin greatly increase the blood volume and may sustain the circulation for a time, but only rarely result in recovery. A marked bleeding tendency is produced by this therapy. Concentrated (25 per cent) bovine albumin solution in equivalent or greater protein content is of no benefit, even if supplemented by saline solution.

Large volume intravenous infusion therapy, using either saline solution alone or albumin in saline solution, is harmful by producing marked edema of tissues, serous effusion, venous distention, and widespread hemorrhage from small vessels.

Pitressin, with or without ergotamine, is of no value. The combination of pitressin with 5 per cent albumin solution is not beneficial. Paredrine causes an elevation of the arterial and venous blood pressure and no improvement in cardiac output. The duration of this effect is limited by the rapid development of unresponsiveness to the drug, and

survival time is not prolonged. Coramine increases skeletal muscle tone, but does not favorably influence the course of events.

The correction of acidosis by the administration of sodium bicarbonate with the initial transfusion does not alter the deteriorating trend of advanced hemorrhagic shock. Sodium succinate is of no benefit in the therapy of advanced hemorrhagic shock. Tuamine, given when the initial transfusion is failing, causes a transitory rise in blood pressure but the effect is brief. Survival time is not prolonged. "Potassium phosphate" intracisternally did not alter the deteriorating trend of hemorrhagic shock and at the same time produced undesirable cerebral excitatory phenomena.

It is the authors' view that advanced shock constitutes a state of progressive deterioration which is not amenable to the types of therapy now available, probably because fundamental biochemical changes have developed as a result of prolonged deficiency of capillary flow. These changes may result from injury predominantly involving one vital organ, such as the liver, or from widespread cellular damage. AUTHORS.

Goldberg, M., and Fine, J.: Traumatic Shock, XI. Intestinal Absorption in Hemorrhagic Shock, J. Clin. Investigation **24:** 445, 1945.

In hemorrhagic shock, the small intestine is progressively deficient in its absorptive capacity for water and isotonic glucose. After transfusion, whether effective or not, some improvement in this function may be observed, but full recovery is not observed within the time interval of these experiments.

The absorption of physiologic saline is not clearly affected by the institution of hemorrhagic shock until the advanced stage of shock is reached. AUTHORS.

Hunter, J. B.: Observations on Ligature of the Patent Ductus Arteriosus. Brit. M. J. **1:** 731, 1945.

Fourteen cases are reported in which ligation of a patent ductus arteriosus has been performed. Twelve cases were uncomplicated by infection, and in only two was endocarditis present. In ten cases the patients were female and the ages varied between 5 and 31 years. In the noncomplicated cases, the patients were all slightly stunted in growth, and the majority were breathless on exertion, but only one showed any signs of cyanosis. It is too soon to see what the ultimate fate of these cases may be. One of the children is now taking up tap dancing as a profession; the young male was accepted by the Service. The others have lost their breathlessness on exertion, and the earlier ones that have been recently reviewed have improved in general health and physique. MCCULLOCH.

Gottschall, R. Y., Laurent, D., DeKruif, P., Simpson, W. M., Kendell, H. W., and Rose, D. L.: The Effect of Artificially Introduced Fever on Humoral Antibodies and on Histamine Intoxication in the Guinea Pig. J. Lab. & Clin. Med. **30:** 563, 1945.

An artificially induced fever temperature of 42.2° C. maintained for thirty minutes has no effect upon the humoral precipitin titer of guinea pigs immunized against horse serum.

The antiprotein titer of sensitized guinea pigs is not significantly altered by a fever temperature of 42.2° C. maintained for sixty minutes when the serum is titrated by a modified Prausnitz-Küstner reaction.

A fever temperature of 42.2° C. maintained for thirty minutes suppresses histamine shock.

In vitro tests with surviving normal intestine exposed to histamine indicate that there is less contraction at 43.3°, 45°, or 46° C. than at 38.8° or 39° C. Response of sensitized intestine to the specific antigen (ovalbumin) is also decreased by temperatures of 43.3°, 45°, or 46° C.

The immediate type of reaction in the guinea pig's skin is suppressed by a fever temperature of 42.2° C. maintained for sixty minutes when the locally sensitized tissue is injected with the specific antigen during hyperpyrexia. AUTHORS.

Book Reviews

A PRIMER OF ELECTROCARDIOGRAPHY: By George Burch, M.D., and Travis Winsor, M.D., Lea & Febiger, Philadelphia, 1945, 215 pages, including table of contents, appendix, and index, 235 engravings.

The title of this book is not entirely adequate, as the work contains much more than on elementary discussion of electrocardiography. The authors obviously have considerable knowledge of the electrical phenomena responsible for the electrocardiogram, and, in this respect, the book is quite different from most available texts on this subject. In the presentation of electrocardiograms related to various cardiac conditions, emphasis is placed upon the reasons for the appearance of characteristic alterations in the tracings. This approach, in the opinion of the reviewer, is so far superior to the purely descriptive method, so often employed, that this book stands nearly in a class by itself.

The first chapter, which occupies approximately one quarter of the book, is largely concerned with a description of the elements which make up the normal electrocardiogram, discussions of the fundamental concepts relating to depolarization and repolarization and the flow of currents in volume conductors, and finally with a presentation of the standard limb leads and the electrical axis viewed from the standpoint of these basic considerations. The remaining four chapters take up important electrocardiographic conditions and include good discussions of precordial leads, the cardiac arrhythmias, and the ventricular gradient.

The changes produced by myocardial infarction are well presented although the reader might find the arguments easier to follow if all the discussions relating to this matter were in one section rather than being distributed in Chapters II, III, and V. Since the basic changes in the QRS complex, RS-T segment, and T wave due to acute injury, myocardial ischemia, and infarction are found in a more or less modified pattern in any lead that is clinically useful but occur in the purest form in precordial (or esophageal) leads, it seems logical to open the discussion of these matters by detailed descriptions of the alterations that are found in leads of the latter type. Changes to be expected in the standard limb leads may then be directly inferred if one remembers that anterior infarcts produce modifications in these leads only when the potential of the left arm is altered in the characteristic manner and posterior infarcts produce them, only when the potential of the leg is similarly changed.

There are not many features of this book with which one can violently disagree, but a few minor criticisms may be justified. The classification of complete bundle branch block into a typical and an atypical variety on the basis of the direction of the T waves is a bit arbitrary. As a matter of fact, activation or depolarization of the ventricles occurs in an identical fashion in the two groups. It is recovery and not activation which is atypical.

In the discussion of the method for taking precordial leads with the use of the central terminal, it is stated (page 112) that, since the sum of all the forces (currents) in a network flowing toward a single point is zero, the central terminal is, for all practical purposes, at zero potential at all times. While we believe the conclusion here to be a true one, unfortunately this question cannot be settled so easily.

It is perhaps to be regretted that no actual electrocardiograms are reproduced in the book. On the other hand, the entire approach is to explain why things occur and not simply to illustrate things that have happened. For this purpose, the numerous well-drawn figures and electrocardiograms serve admirably. As the authors remark in the preface, other books are available in which many well-reproduced tracings may be found. In the opinion of the reviewer, there are few other books that can offer as much as this one to the student really interested in the fundamental aspects of electrocardiography.

F. D. JOHNSTON.

Announcement

Due to conditions beyond the control of the editors and publishers, several issues of the JOURNAL are printed on an inferior grade of paper. Just as soon as the standard good grade of paper is available, its use will be resumed.

American Heart Association, Inc.

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